

24th Annual International Symposium
on
Man and His Environment in Health and Disease

Special Focus

**Environmental Aspects and Treatment of Reversibility of Chronic Disease
and Hypersensitivity, Sulfur Compounds, Mycotoxins and Hormetic Effect
of Chemical Sensitivity and Chronic Degenerative Disease**

Sponsored by
American Environmental Health Foundation
and
University of North Texas Health Science Center

Physician Accreditation/Credit:

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education and the American Environmental Health Foundation. The University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education is accredited by the ACCME to provide continuing medical education for physicians.

The University of North Texas Health Science Center at Fort Worth is accredited by the American Osteopathic Association to award continuing medical education to physicians.

The University of North Texas Health Science Center at Fort Worth designates this educational activity for a maximum of 24 Category 1 credits toward the AMA Physician's Recognition Award. Each physician should claim only those credits that he/she actually spent in the activity.

The University of North Texas Health Science Center anticipates this program for 24 hours in Category 2A CME credit hours, pending approval from the American Osteopathic Association.

Nursing Accreditation/Credit:

University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education, Provider #02588A, is approved provider of continuing nursing education by the Texas Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation. This activity meets Type 1 criteria for mandatory continuing education requirements toward relicensure as established by the Board of Nurse Examiners for the State of Texas. This activity is approved for 28.8 Contact Hours.

To receive a certificate of successful completion, participants must attend the activity in its entirety and complete and return the activity evaluation credit request form.

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FINANCIAL CONSIDERATION

AEHF is a nonprofit organization that was founded in 1975 to provide education and research into Environmental Medicine. This year's Symposium is our 24th Annual International Symposium and is our major vehicle for educating the medical professional.

Funding for the symposium is provided by registration fees from physicians and exhibitors. Proceeds from the AEHF store cover the shortfall between registration fees and expenses for the conference. AEHF does not receive grants or any outside financial support for our education. Donations are accepted and used toward research into environmental medicine.

INTRODUCTION

SYMPOSIUM PURPOSE

Since 1981, the International Symposium has been recognized as one of the most advanced medical forums in the world addressing the research and treatment of environmental effects on health and disease. The 2006 conference will focus on “Environmental Aspects and Treatment of Reversibility of Chronic Disease and Hypersensitivity, Sulfur Compounds, Mycotoxins and Hormetic Effect of Chemical Sensitivity and Chronic Degenerative Disease.” This Conference presents the most current information available while providing guidelines to identify, diagnose, treat and to prevent environmentally triggered responses in the body.

GOALS OF THE MEETING

- ! To provide new insights into the mechanisms and the environmental causes behind many problems seen by the physician.
- ! To present new diagnostic and treatment modalities to help improve the quality of care for your complex patients.
- ! To provide concepts, tools that will enhance the physicians practice.

OBJECTIVES OF THE MEETING

- ! Improve the outcome of treating patients with Chronic Disease and Hypersensitivity.
- ! Use new concepts and treatments to help better diagnose and manage many patients with Chronic Disease and Hypersensitivity.
- ! Apply the concepts of this conference to your practice by using nutrition and environmental manipulation for the treatment of Chronic Disease and Hypersensitivity.
- ! Use the information presented to enhance the effectiveness, cost-efficiency, and competitiveness in relation to Chronic Disease and Hypersensitivity.

INTENDED AUDIENCE

M.D.=s, D.O.=s, D.D.S.’s medical students, nurses, nutritionists and other health professionals interested in the concepts and practice of Environmental Medicine, Occupational Medicine and Toxicology.

EDUCATIONAL FORMATS

- ✂ Plenary
- ✂ Panels Discussions
- ✂ Case Studies
- ✂ Question & Answer Sessions.

CONFERENCE FORMAT

The AEHF Committee has selected some of the leading experts in the fields of chronic disease, nutrition and chemical sensitivity.

Each speaker=s presentation will last approximately 20 minutes and will be followed by a 10 minute question and answer session. All speakers are encouraged to use any and all appropriate audio/visual aids. (A brief outline of the speech is included in this booklet.)

GIVEN IN COOPERATION

William J. Rea, M.D., F.A.C.S.

Symposium Chairman,
American Environmental Health Foundation,
Environmental Health Center - Dallas,
Dallas, Texas

Bertie B. Griffiths, Ph.D.,

Environmental Health Center - Dallas
Dallas, Texas

Kaye H. Kilburn, M. D.

University of Southern California Medical Center
Keck School of Medicine
Los Angeles, CA

William J. Meggs, M.D., Ph.D.

Brody School of Medicine, East Carolina University
Department of Emergency Medicine
Greenville, NC

24th ANNUAL INTERNATIONAL SYMPOSIUM

ON MAN & HIS ENVIRONMENT

Schedule

Thursday, June 8, 2006

7:00 a.m. REGISTRATION

9:00 WELCOME/MODERATOR: William J. Rea, M.D. and Kalpana Patel, M.D.

9:10 Doug Seba, Ph.D., "Update 2006: Hormesis and the Environment"

9:30 Q & A

9:40 Jonathan Fox, M.D., "Measuring Adaptation in the Chemically Sensitive Individual"

10:00 Q&A

10:00 BREAK

10:30 William J. Rea, M.D., "Diagnosis of Chronic Hypersensitivity and Disease: The Hormetic Effects"

10:50 Q & A

11:00 Kaye H. Kilburn, M.D., "Human Costs of Rotten Egg Gas (REG) often an Insidious Poison"

11:20 Q & A

11:30 William J. Meggs, M.D., Ph.D., "The Obesity Epidemic and Environmental Toxins"

11:50 Q & A

12:00 LUNCHEON IN SPURS RESTRAUNT

1:30 **MODERATOR:** Tipu Sultan, M.D.

1:30 Kenneth Fine, M.D., "Diagnosis of Gluten Sensitivity Using Fecal Testing"

1:50 Q & A

2:00 Joaquim Fernandez Sola, M.D., "Chronic Fatigue Syndrome Induced by Toxic Agents"

2:20 Q & A

2:30 Richard Jaeckle, M.D., "Targeted Amino Acid Therapy (TAAT) Support in Chronic Illness"

2:50 Q & A

3:00 BREAK

3:30 Panel Discussion/Case Studies: Allan D. Lieberman, M.D. and William J. Rea, M.D., "Hydraulic Fluid Toxicity in a Surgical Suite"

5:00 AJOURN

THURSDAY, JUNE 8, 2006

ABSTRACTS

AND

HANDOUTS

Objectives & Notes

Doug Seba, Ph.D.

Date of talk: Thursday, June 8, 2006, 9:10am

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Alexandria, VA 22313

Phone: 703/949-1055

Training:

Current Job Description:	Independent Marine Scientist
Current Faculty Appointments:	None
Medical School/ University Attended	University of Miami, M.S., Ph.D.
Other Information: (including titles of books or articles you have recently written):	Over 45 years experience with chemicals and the environment.

SPEECH TITLE: "Update 2006: Hormesis and the Environment"

At the end of this Presentation, the participant should be able to:

1. Understand multiple definitions of hormesis and have several source information resources.
2. Realize the many limitations of the hormetic effect in environmental medicine.
3. Appreciate the scope of politics, policy, and science underlying the everyday application of the hormetic effect.

The above information was provided by the Speaker.

Update 2006: Hormesis and the Environment

Douglas B. Seba, Ph.D.

Hormesis is increasingly being invoked in wide variety of toxicology phenomenon to explain paradoxical effects wherein low doses of harmful substances are beneficial in contrast to their adverse effects at higher doses. This review paper will examine the ever-expanding definitions of hormetic effects to environmental medicine. An understanding of politics, policy, and science behind hormesis is necessary to know when or if its application is appropriate.

This paper will also update ongoing research of the author of interest to attendees including molds, atmospheric dust, endocrine disruptors and comparative hormetic effects.

Overhead 2

HORMESIS

IS A LITTLE POISON GOOD FOR YOU?

OR

MUCH ADO ABOUT NOTHING?

ANSWER

IT DEPENDS ON YOUR DEFINITION

Overhead 3

www.HormesisSociety.org

NONLINEARITY IN BIOLOGY, TOXICOLOGY, AND MEDICINE

PHYSIOLOGY	BIOCHEMISTRY
MEDICINE	TOXICOLOGY
PHARMACOLOGY	MOLECULAR
	BIOLOGY
RADIATION BIOLOGY	PLANT BIOLOGY
EXPERIMENTAL	ENVIRONMENTAL
PSYCHOLOGY	SCIENCES

LOCATED IN THE SCHOOL OF PUBLIC HEALTH, UNIVERSITY OF MASSACHUSETTS

HISTORICAL FIGURES:

HUGO SCHULZ
FERNANDO HUEPPE
CHESTER SOUTHAM & JOHN EHRLICH

HORMESIS: A DOSE-RESPONSE PHENOMENON CHARACTERIZED BY LOW-DOSE STIMULATION AND HIGH-DOSE INHIBITION.

Overhead 4

**BELLE: A STRATEGY TO ENCOURAGE
THE ASSESSMENT OF THE
BIOLOGICAL EFFECTS OF
LOW-LEVEL EXPOSURES TO
CHEMICAL AGENTS AND
RADIOACTIVITY**

ADVISORY COMMITTEE

CHAIRMAN: EDWARD J. CALABRESE, Ph.D.

COMMITTEE MEMBERS INCLUDE MANY

ENERGY AND CHEMICAL COMPANIES

Overhead 5

**DOSE-RESPONSE: ASSESSING THE
NATURE, MECHANISMS, AND
IMPLICATIONS OF DOSE-RESPONSE
RELATIONSHIPS**

**THRESHOLD ADAPTIVE
BIDIRECTIONAL BIPHASIC
HORMETIC U/J-SHAPED
NON-MONOTONIC
PARADOXICAL
YERKES-DODSON LAW
SUBSIDY-STRESS GRADIENT
REVERSE DOSE-RESPONSES**

Overhead 6

PROTECTIVE BYSTANDER EFFECTS FOLLOWING LOW DOSE IONIZING RADIATION

PROLONGATION OF LIFE SPAN OF DISEASE MODEL MICE BY LOW DOSE IRRADIATION

**SMOKING AND HORMESIS AS CONFOUNDING FACTORS IN RADIATION PULMONARY
CARCINOGENESIS**

Overhead 7

GENERAL TOXICOLOGY

WILDLIFE EFFECTS SIMILAR TO HUMAN

ENDOCRINE DISRUPTERS OPPOSITE OF HORMESIS EFFECTS (LIKE VITAMINS)

STUDIES USUALLY DONE GENETICALLY UNIFORM ANIMALS

GENERALLY IGNORE FACT THAT GENE EXPRESSION IS NOT FIXED

MOST LOW-DOSE STUDIES ON AQUATIC ORGANISMS

GENERALLY IGNORE EFFECTS OF SPECIES SPECIFIC HORMONES (VITELLOGENIN AND TRENBOLONE)

GENERALLY IGNORE EFFECTS OF AGE DIFFERENCES ON DETOXIFICATION ENZYMES (YOUNG AND OLD)

GENERALLY IGNORE ENVIRONMENTAL EFFECTS OF SENSITIVITY

Overhead 8

LOW DOSE SAFETY

REGULATORY TOXICOLOGY

RESEARCH TOXICOLOGY, NON-GUIDELINE

TIMING - WOMB, INFANT, OLD AGE

IMMUNE STATUS

GENETIC VARIATION

BIPHASIC DOSE RESPONSE

DISEASE RISK

DELAYED REACTIONS

NOEL'S AND LOW DOSE SAFETY A REGULATORY ARTIFACT - THERE ARE NO WOMB TO TOMB STUDIES

Overhead 9

DEFINING HORMESIS:

HORMESIS SHOULD BE CONSIDERED AN ADAPTIVE RESPONSE CHARACTERIZED BY BIPHASIC DOSE RESPONSES OF GENERALLY SIMILAR QUANTITATIVE FEATURES WITH RESPECT TO AMPLITUDE AND RANGE OF THE STIMULATORY RESPONSE THAT ARE EITHER DIRECTLY INDUCED OR THE RESULT OF COMPENSATORY BIOLOGICAL PROCESSES FOLLOWING AN INITIAL DISRUPTION IN HOMEOSTASIS.

Overhead 10

Google: homopathic OR homeopathic

10,200,000 results

hormesis

139,000 results

hormesis Calabrese

1,715 results

Overhead 11

EPA hormesis search results: 89 documents

**Draft Science Advisory Board document
December 27, 2005 Do not Cite or Quote.
Not approved by the Chartered SAB.
Does not represent Agency policy.**

in document on Arsenic it is mentioned that you should not dismiss the possibilities of hormesis effects...essentiality to humans...

Overhead 12

EPA SCIENCE INVENTORY #1 1/5/06

AUTHORS: KT KITCHIN & W DRANE

A CRITIQUE OF THE USE OF HORMESIS IN RISK ASSESSMENT

“...SEVERE PROBLEMS ..LIMITATIONS WITH HORMESIS...”

- A) unknown prevalence of hormetic dose-response curves**
- B) random chance occurrence of hormesis and the shortage of data on the repeatability of hormesis**
- C) unknown degree of generalizability of hormesis**

Overhead 13

EPA SCIENCE INVENTORY #1 1/5/06

D) There are dose response curves that are not hormetic, therefore hormesis cannot be universally generalized

E) Problems of post hoc rather than a priori hypothesis testing

F) A possible large problem of ‘false positive’ hormetic data sets which have not been extensively replicated

G) The ‘mechanism of hormesis’ is not understood at a rigorous scientific level

H) In some cases hormesis may merely be the overall sum of many different mechanisms and many different dose-response curves - some beneficial and some toxic

Overhead 14

ProQuest Search yields 25 Scholarly Journals publishing hormesis articles

IngentaConnect yielded the most recent article (as of 04/04/06) using hormesis

“Toward a population ecology of stressed environments: the effects of zinc on the springtail *Folsomia candida*”

Authors: Noel, Helen L., *et.al.*

Overhead 15

Chemical & Engineering News

Government & Policy Section Article

April 5, 2004, 5pp.

Low-Dose Effects: Debate expands on how to extrapolate data from high-dose test for environmental contaminants.

Overhead 16

“Hormesis’ - An Inappropriate Extrapolation from the Specific to the Universal.”

Authors: D. Axelrod, *et.al.*

International Journal Occupational and Environmental Health 2004; 10:335-339

Overhead 17

**The National Academies of Science/
National Research Council**

Board on Environmental Studies and Toxicology

Committee on Toxicity Testing and Assessment of Environmental Agents

Report due summer, 2006

Overhead 18

**The National Academies of Science/
National Research Council**

Nuclear and Radiation Studies Board

Committee of Biological Effects of Ionizing Radiation

BEIR VII, 5 year study, released 06/30/05

“Health risks from exposure to low-levels of ionizing radiation”

“...data supports a linear-no-threshold risk model...”

Overhead 19

www.tera.org

TERA: Toxicology Excellence for Risk Assessment

Non-profit with mission to protect public health by developing and communicating risk assessment information, sponsoring peer reviews and consultations, improving risk methods through research, and educating the public on risk assessment issues.

Overhead 20

TIACT: The Texas Institute for Advancement of Chemical Technology

**“A nonprofit, charitable corporation
dedicated to the advancement of chemical technology through an informed public.”**

Located at Texas A&M University, College Station, Texas.

<http://cheweb.tamu.edu/tiact>

INSIGHTS: Chemical Hormesis: Beneficial Effects at Low Exposures, Adverse Effects at High Exposures – 1998

Overhead 21

“The scientific acceptance of hormesis with its possible benefits at low-level exposure could come at no better time than the present when environmentalists and others are calling for bans on more and more chemicals, such as chlorinated hydrocarbons to prevent low-level exposures. Furthermore, the low-exposure paradigm would make it possible for society to enjoy, safely, the benefits of many chemicals that have been banned in the past or could be banned in the future”

Page 1 of the executive summary

Overhead 22

Compensation for Sick Nuclear Workers

Congress passed law in 2001

\$150,000 plus medical benefits

About 73,000 workers or their survivors have filed claims for cancer

National Institute for Occupational Safety and Health oversees contractor, the Oak Ridge Associated Universities

How much radiation were they exposed to and how harmful was it?

Overhead 23

www.ombwatch.org

“Democracies die behind closed doors”

Court rejects data quality act case brought by industry

March 6, 2006, Fourth Circuit dismissed

Judges found:

A) Act does not allow for judicial review

B) Plaintiffs had not shown injury

What happens next?

Overhead 24

Federal Judicial Center is a research and education agency of the federal judicial system located in Washington, D.C.

A survey found that judges in dealing with *Daubert* only 5% clearly understand falsifiability and only 4% clearly understand error rate.

www.savekevincooper.org

Did blood on shirt contain EDTA?

Current Health and Human Services appropriation bill has amendment prohibiting political tests for science advisory committees, the first time in our 230-year history.

Overhead 25

EPA RULE FOR HUMAN TESTING

Issued January 26, 2006

Lawsuits filed February 23, 2006

Appears to allow testing on pregnant women and children if not to be used in rule making and allows dosing up to tolerance levels.

Pesticide Action Network North America

Overhead 26

Terbutaline sensitizes brain to pesticide

1 million pre-term labor drug patients/yr

Common pesticide Chlorpyrifos/Dursban

Rats exposed at 2-4 days (early 3rd trimester) to terbutaline, then Dursban days 11-14. Loss of brain cells/nerve projections persisted into adulthood.

Theodore Slotkin, Duke University, Durham, North Carolina

Overhead 27

Bacteroides DNA sequencing antibiotics

Bacteroides constitutes 25% gut flora.

Tetracycline resistance under 25% in 1970 samples, increased to over 85% by 1990. This resistance passed to other gut bacteria by both conjugation and transformation.

“Viewed in this way, the human colon is the bacterial equivalent of eBay”

Abigail Salyers, U. Illinois at Urbana

Do not confuse contagion theory with hormesis. Hormesis is about chemicals.

Do not confuse hygiene hypothesis with hormesis. Hormesis is about chemicals.

Overhead 28

Radicals (ROS) tell mice up from down

ROS make vesicles permeable to calcium to form otolith. Mutant ‘head-slant’ mice block noxol, a regulatory protein .

Botond Banfi, U. Iowa, Iowa City, Iowa

Overhead 29

SO WHERE ARE WE?

- 1) Aspirin
- 2) Coffee
- 3) Cadmium
- 4) X-rays
- 5) Pesticides
- 6) Ground/Kindling/Frequency
Masking/Adaptation/MCS

**New branch of pharmacology
or
toxicology epiphenomenon?**

Overhead 30

Codex Alimentarius/WTO

Food & Agricultural Organization/WTO

“A model for establishing upper levels for nutrients and related substances”

The Nutrient Risk Assessment Project treats nutrients the same as industrial toxins, *i.e.*, low good, high bad.

GAO-01-1139T (January 2006) Health Products for Seniors: Potential Harm From ‘Anti-Aging’ Products

...particularly risky...underlying disease...medically inadvisable supplements...may have serious health consequences...

Overhead 31

STATUS INCONSISTENCY THEORY AND FLYING SAUCER SIGHTINGS

“This sociological theory is further validated through analysis of a national survey of sighters.”

Donald I. Warren, Science, 06/11/1970 Vol. 170:pp 599-603.

Overhead 32

SPIRITES

Davis Sentman, U. Alaska

SELF-ASSEMBLING HALOALKANES

John C. Polanyi, *et.al.*, U. Toronto, Canada

SELF-ASSEMBLING HYALURONAN

Raymond E. Turner, U. Massachusetts, Cambridge

Overhead 33

Rice blight is caused by fugal symbiosis

Fungal toxin Rhizoxin is not produced by *Rhizopus* itself but by symbiont bacterium. This is a complex and unprecedented alliance.

Christian Hertwerk, Institute for Natural Products Research & Infection Biology, Jena, Germany

Overhead 34

Fungi defense

The first time a defensin antibiotic has been found in a fungus. The peptide antibiotic, Plectasin, made by the fungus *Pseudoplectania nigrella*, is particularly effective against *Streptococcus pneumoniae*.

Hans-Henrik Kristensen, Novozymes, Bagsvaerd, Denmark

Overhead 35

THE DISAPPEARING MALE BABY

Chippewa Indian Reservation, Samia, Ontario, Canada

106 to 100 ratio has fallen to 105 to 100 throughout industrial world last decade.

In Samia, it is 35 to 100, where land is now surrounded by extensive petrochemical complexes.

Dioxin, Seveso, Italy, ratio 50 to 100.

Ada Lockridge, U. Ottawa, Canada

Number of American males still living at home age 20-35 has doubled last decade, no change in number of females.

Shanna Swan, U. Rochester, New York

Overhead 36

NATIONAL PESTICIDE SURVEY

USGS study of 51 major US river basins from 1992 to 2001 and groundwater

40 of 100 pesticides were found most of the time in water and sediments at concentrations that could affect aquatic life or fish-eating wildlife.

<http://water.usgs.gov/nawqa>

Overhead 37

GUPPY GONOPODIAL THRUSTS GONE WITH DURSBAN

Guppies (*Poecilia reticulata*) exposed to chlorpyrifos at 0.002micrograms/liter had 16 thrusts/hr vs. 44 for controls.

Live birth reduced from 27 to 8 per female.

Offspring survival at 14 days reduced from 94% to 47%.

P.M.C.S. De Silva, U. Bergen, Bergen, Norway

Overhead 38

HUMAN MALE INFERTILITY FROM CHLORPYRIFOS

268 men, infertility clinic, 2000-2003.

Level of chlorpyrifos metabolite (TCPY) inversely related to sperm count, DNA damage, testosterone levels, and free androgen index.

John D. Meeker, U. Michigan, Ann Arbor

Overhead 39

CARBAMATE EXPOSURE DURING PREGNANCY/EARLY CHILDHOOD DOUBLES RISK OF LEUKEMIA

Common carbamate include Carbaryl, Carbofuran, and Carbosulfan.

280 childhood leukemia cases, 288 controls

Florence Menegaux, INSERM, Villejuif, France

Overhead 39

AFRICAN DUST

Affects thunderstorms in Florida - smaller, more intense, more heat, less rain

Susan van den Heever, Colorado State U., Fort Collins, Colorado

Aerosol cooling of Earth greater than expected - from 0.2 to 0.8W/square meter, green houses gases about 2.4W/sq. meter.

Nicolas Beilouin, U. K. Meteorological (Met), London, England

African dust increases paediatric asthma

K. Gyan, U. West Indies, Trinidad, W. I.

Miami Idiopathic Pulmonary Fibrosis Symposium - www.ipfmiami.org

Overhead 40

SCIENCE HAIKU

**TO STUDY WHAT YOU
INSTINCTIVELY BELIEVE IS
THE BASIS OF SCIENCE**

Objectives & Notes

Jonathan Fox, M.D.

Date of talk: Thursday, June 8, 2006, 9:40am

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Fall River, Nova Scotia B2T 1K6
Canada

Phone: 902/860-1890
Fax: 902/860-2046
Email: jonathan.fox@cdha.nshealth.ca

Training:

Current Job Description:	Physician – patient care Nova Scotia Environmental Health Centre
Current Faculty Appointments:	Department of Family Medicine – Dalhousie University
Medical School/ University Attended	Dalhousie
Internship:	University of Ottawa
Residency:	University of Ottawa – Family Medicine
Board Certifications:	Family Medicine

SPEECH TITLE: “Adaptation in Individuals with Multiple Chemical Sensitivity”

At the end of this Presentation, the participant should be able to:

1. Recognize that individuals with MCS have a change in their adaptation.
2. There is no single pattern for all patients.
3. MCS patients display non-specific heightened reactivity.

The above information was provided by the Speaker.

Adaptation in Individuals with Multiple Chemical Sensitivity

Jonathan Fox¹, Tara Sampalli¹ and Roy Fox¹

¹ Nova Scotia Environmental Health Centre

In a study conducted at the Nova Scotia Environmental Health Centre with 31 individuals with MCS and 17 controls, the response to environmental triggers such as variation in noise levels and exposures to low levels of chemicals was tested. All subjects were given up to 4 sessions to adapt to the baseline experimental changes before challenging them to an everyday chemical in a randomized sequence. Skin conductance was the main indicator of adaptation and reactivity to chemicals. The baseline experimental variations such as the sound of the exhaust fan, sound of the booth door opening and closing and subjects wearing nose plugs remained the same every session in order to allow the subjects to adapt to them. Of the 31 MCS subjects, 2 did not adapt past 4 sessions and one subject discontinued after the first session in the booth. The 28 MCS subjects that completed the study were physiologically different from the controls in their adaptation to the baseline experimental protocols and in their reactivity to chemicals. In the control group, 86% adapted in one session to the baseline experimental protocols and 14% adapted in two sessions. In the MCS group, 54% adapted in 2 sessions, 18% in 3 sessions and 24% in 4 sessions with only 4% adapting in the first session. Within the MCS group, there were identifiable variations in the adaptation process. The 2 subjects that did not adapt past the 4 sessions displayed higher tonic skin conductance levels with non-specific reactivity during the sessions and continued to maintain the high tonic baseline levels and non-specific reactivity past 4 sessions. Of the 24% that adapted in 4 sessions, 11% showed a gradual reduction in their high tonic levels of skin conductance by the 4th session with the non-specific reactivity changing to specific reactivity in the process of adaptation. Another 13% took up to 4 sessions to adapt with specific reactivity to the stimulus and lower tonic baseline levels of skin conductance. A one-year follow-up testing revealed changes in adaptation and reactivity in two of the subjects from the study. One of the subjects tested in follow-up had not adapted past 4 sessions and the other subject had taken up to 4 sessions with elevated tonic levels gradually reducing by the 4th session. The two MCS subjects adapted more rapidly to baseline experimental stimuli with lower tonic levels of skin conductance. The two subjects who were new patients of the Centre at the time of the research study had completed several treatment modalities in the one-year follow up testing.

REFERENCE

1. Bartha L, Baumzweiger W, Buscher DS. Multiple chemical sensitivity: a 1999 consensus. *Arch Environ Health*. 1999;54:147-149.
2. Bensafi, C, Rouby, V, Farget, B, Bertrand, M, Vigouroux and A. Holley. Autonomic Nervous System Responses to Odour: the Role of Pleasantness and Arousal; 2002; *Chem. Senses* 27: 703-709.
3. Greenwald, M.K., Cook, E.W. and Lang, P.J. Affective judgment and psychophysiological response: dimensional covariation in the evaluation of pictorial stimuli. *J. Psychophysiol.*, 1989, 3,51 -64.
4. Joffres MR, Sampalli T, Fox RA . Physiologic and Symptomatic Responses to Low-Level Substances in Individuals with and without Chemical Sensitivities: A Randomized Controlled Blinded Pilot Booth Study, *Environmental Health Perspectives*, September 2005, 113(9): 1178 - 83.
5. Lang, P.J., Bradley, M.M., and Cuthbert, B.N. Emotion and motivation: measuring affective perception. *J. Clin. Neurophysiol.*,1998 (b);15 , 397-408.
6. Lim CL, Rennie C, Barry RJ, Bahramali H, Lazzaro I, Manor B, et al. Decomposing skin conductance into tonic and phasic components. *Int J Psychophysiol*. 1997;25:97-109.

Objectives & Notes

William J. Rea, M.D.

Date of talk: Thursday, June 8, 2006, 10:30am

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Training:

Current Job Description:	M.D., President, Environmental Health Center – Dallas
Current Faculty Appointments:	Capital University of Integrative Medicine
Medical School/ University Attended	Ohio State University College of Medicine, Columbus, OH
Internship:	Parkland Memorial Hospital, Dallas, TX
Residency:	University of Texas Southwestern Medical School
Board Certifications:	American Board of Surgery, American Board of Thoracic Surgery, American Board of Environmental Medicine
Other Information: (including titles of books or articles you have recently written):	Optimum Environments for Optimum Health and Creativity (book); Averse Health Effects of Indoor Molds.

SPEECH TITLE: “**Diagnosis of Chronic Hypersensitivity and Disease: The Hormetic Effects**”

At the end of this Presentation, the participant should be able to:

1. To understand hormesis.
2. To understand how hormesis works.
3. Apply the hormetic effects to the diagnosis of chronic hypersensitivity.

The above information was provided by the Speaker.

DIAGNOSIS OF CHRONIC HYPERSENSITIVITY AND DISEASE: THE HORMETIC EFFECTS

William J. Rea, M.D., F.A.S.C., FA.A.E.M.

The diagnosis of chemical sensitivity and chronic degenerative disease takes on a whole new point of view when one introduces the hormetic principle in addition to the linear threshold dose effect. With hormesis, there are “j” or “u” curves paralleling stimulating effects usually at low doses and inhibitory effects usually at high doses; therefore, the same chemical at different doses can give the opposite effect. Using this principle can change our whole perception of the effects of different incitants.

Conclusion: The recognition of this principle can simplify diagnostic capabilities as well as complicate them due to the complex nature of multiple incitants occurring almost simultaneously.

References:

Calabrese, E.J. and Baldwin, L.A., Special Issue on hormesis, *Hum. Exp. Toxicol.*, 19(1), 2-97, 2000.

Calabrese, E.J. and Baldwin, L.A., A quantitatively based methodology for the evaluation of chemical hormesis, *Hum. Ecol. Risk Assess.*, 3(4), 545-554, 1997.

Objectives & Notes

Kaye H. Kilburn, M.D.

Date of talk: Thursday, June 8, 2006, 11:00am

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Training:

Current Job Description:	Professor
Current Faculty Appointments:	Ralph Edgington Professor of Internal Medicine at University of Southern California Keck School of Medicine
Medical School/ University Attended	University of Utah College of Medicine
Internship:	Western Reserve University Hospital, Cleveland, Ohio
Residency:	Utah, Duke, University of London
Board Certifications:	American Board of Internal Medicine, American Board of Preventive Medicine - Occupational Health
Other Information: (including titles of books or articles you have recently written):	Books: Chemical Brain Injury, Endangered Brains, Molds and Mycotoxins. Archives of Environmental Health: Onboard insecticide use of flight attendants, Affects of chlorine and other chemicals byproducts, including mold and mold products, in brain and lung performance. Archives of Environmental Health

SPEECH TITLE: **“Human Costs of Rotten Egg Gas (REG) often an Insidious Poison”**

At the end of this Presentation, the participant should be able to:

1. Understand how H₂S exposures occur and where
2. Associate neurobehavioral impairment with headache, memory loss and trouble concentrating
3. See what must be done to prevent exposures to H₂S

The above information was provided by the Speaker.

HUMAN COSTS OF ROTTEN EGG GAS (REG) OFTEN AN INSIDIOUS POISON

Running title: Hydrogen sulfide poisoning at home

¹**Kaye H. Kilburn, M.D.**

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Background Fatalities from REG have described since 1845 but community poisoning is new. We describe families exposed to REG from Confined Animal Feeding Operation (CAFO) lagoons in Ohio and in Lovington, a community in New Mexico. The Ohio objective was to determine whether neighbors around manure lagoons and massive hog confinement buildings who complained of offensive odors and symptoms had impaired brain and lung performance. In Southeastern New Mexico, 5 sewer workers and 3 of their wives had shown neurobehavioral impairment associated with hydrogen sulfide (H₂S). Wives were more impaired than the workers suggesting environmental exposure so we tested other people in the town. Major sources of H₂S were the municipal sewer treatment plant, oil and a gas refinery, and a cheese factory.

Methods We compared neighbors of hog lagoons in Ohio to people living beyond 3 kilometers from them and to controls in a nearby state by testing neurophysiological, cognitive, recall and memory functions, and pulmonary performance. The same 24 neurobehavioral functions were compared in 26 Lovington adults including the 8 seen initially, 12 people from Tatum, NM, 20 miles north, and 11 from Artesia, NM, 60 miles west, and 42 unexposed Arizona people. The Lovington adults were known to have exposure to hydrogen sulfide, while the residents of Tatum and Artesia were thought to have little hydrogen sulfide exposure, although they lived in the “oil patch” of south eastern New Mexico. The 3 groups completed spirometry, a Profile of Mood States, medical and neurological questionnaires including chemical exposures and the frequencies of 35 symptoms. Measurements in both studies were of balance, reaction time, color discrimination, blink reflex, visual fields, grip strength, hearing, and vibration. Subjects were also tested for cognition, verbal recall, memory, attention, concentration, coordination, and finger tip number writing errors (FTNWE). Means of observed measurements were compared by analysis of variance for exposed and unexposed groups after they had been adjusted for effects of age, gender, educational level, height and weight. We also compared groups for numbers of abnormalities defined by scores more than 1.5 standard deviations from predicted values (that were based on referent populations unexposed to chemicals).

Results The 25 Ohio CAFO exposed subjects averaged 4.3 neurobehavioral abnormalities, significantly different from 2.5 for local controls and 2.3 for regional Tennessee controls.

Ages and educational attainment of the groups were not significantly different. Their mean forced vital capacity and expiratory volume in 1 sec. were reduced significantly compared to regional controls but not local neighbors. Lovington people averaged 10.8 abnormalities compared to 4.7 in Tatum and Artesia combined and 2.0 in Arizona unexposed people. The Lovington people were impaired for simple and choice reaction times, balance with eyes open and with eyes closed, visual field score, and hearing and grip strength each on left and right sides. Also all 12 psychological functions were impaired: culture fair, digit symbol substitution, vocabulary, verbal recall, peg placement, trail making A and B, FTNWE, information, picture completion, and similarities. Assessment, by prevalence of abnormalities showed 50% of the Lovington adults had abnormalities for more than 17 neurobehavioral tests, a high total.

Conclusions Gases from hog enclosures and manure lagoon impaired neurobehavioral and pulmonary functions in neighbors and somewhat less impaired neurobehavioral and pulmonary functions in nearby less exposed people who were not controls. REG hydrogen sulfide should be abated for people living near lagoons.

Community exposures to (ambient) hydrogen sulfide from multiple sources were associated with greatly impaired neurobehavioral functions similar to occupational ones, but more prevalent. People in previously studied communities with less effect shared a single hydrogen sulfide source such as an oil refinery, hog husbandry buildings and manure ponds. To plan a human dose response curve is unethical but these associations suggest total avoidance is the path to prevention. The nose knows.

Objectives & Notes

William J. Meggs, M.D., Ph.D.

Date of talk: Thursday, June 8, 2006, 11:30am

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Training:

Current Job Description:	Physician and Research Scientist
Current Faculty Appointments:	Professor, Brody School of Medicine
Medical School/ University Attended	University of Miami, Miami, Florida
Internship:	University of Rochester
Residency:	University of Rochester
Board Certifications:	Medical Toxicology, Allergy and Immunology, Internal Medicine, Emergency Medicine
Other Information: (including titles of books or articles you have recently written):	Book: The Inflammation Cure, McGraw-Hill (2003)/ Fallacies in Refutation of Causality, published in Clinical Toxicology, 2005. Sustained oxygenation without ventilation, published in Amer. Journal Emergency Medicine in 2005. Pressure Immobilization Bandages delay of toxicity of coral snake bites, published in Annuals of Emergency Medicine, 2005.

SPEECH TITLE: "The Obesity Epidemic and Environmental Toxins"

At the end of this Presentation, the participant should be able to:

1. To know the epidemiology of obesity in the USA
2. To know the possible causes of the obesity epidemic
3. To understand the hypothesis that environmental chemicals are responsible for the obesity epidemic.

The above information was provided by the Speaker.

“The Obesity Epidemic and Environmental Toxins”

William J. Meggs, M.D., Ph.D.

There has been a remarkable increase in obesity in the USA over a very short time period. The most prevailing belief that this epidemic is due to sedentary lifestyles and poor nutrition has been challenged. An alternative explanation is that environmental chemicals induce weight gain through a variety of mechanisms, including chemicals that induce obesity has endocrine disrupters. Data will be presented that suggests that chronic low-dose exposures to organophosphate insecticides can induce obesity. Candidate chemicals for playing a role in the obesity epidemic will be presented.

References:

Baillie-Hamilton PF. Chemical toxins: a hypothesis to explain the global obesity epidemic. *J Altern Complement Med.* 2002 Apr;8(2):185-92.

CDC at www.cdc.gov/nccdphp/dnpa/obesity//consequences.htm

Centers for Diseases Control. Third national report on human exposure to environmental chemicals. National Center for Environmental Health report NCEH No. 050570, Atlanta, July 2005.

National task Force on the Prevention and Treatment Of obesity. *Arch Internal Medicine* 2000;160:898-904.

Flegal KM, et al. excess deaths associated with underweight, overweight, & obesity. *JAMA* 2005;293:1861-1867.

Flegal KM et al. Prevalence & trends in obesity among US adults, 1999-2000. *JAMA* 2002;288:1723-1727.

Korner1 J, Aronne LJ. The emerging science of body weight regulation and its impact on obesity treatment *J. Clin. Invest.* 111:565-570 (2003).

Ogden CL et al. Prevalence of overweight & Obesity in US *JAMA* 2006;295:1549-1555

Objectives & Notes

Kenneth Fine, M.D.

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Date of talk: Thursday, June 8, 2006, 1:30pm

Phone: 972/686-6869

Email: info@intestinalhealth.org

Training:

Current Job Description:	Founder/Director Intestinal Health Institute and Enterolab.com Reference Laboratory
Current Faculty Appointments:	Director, Intestinal Health Institute
Medical School/ University Attended	University of Missouri – Kansas City, School of Medicine
Internship:	Baylor University Medical Center, Dallas
Residency:	Baylor University Medical Center, Dallas
Board Certifications:	Internal Medicine, Gastroenterology
Other Information: (including titles of books or articles you have recently written):	Numerous scientific publications on the topics of gluten sensitivity, microscopic colitis, and chronic diarrhea, intestinal physiology.

SPEECH TITLE: “**Diagnosis of Gluten Sensitivity Using Fecal Testing**”

At the end of this Presentation, the participant should be able to:

1. Understand the clinical spectrum of gluten sensitivity vs. celiac disease
2. Realize the shortcomings of blood tests and biopsies for diagnosing gluten sensitivity.
3. How gluten sensitivity stool testing has overcome these problems, and have revealed the prevalence of gluten sensitivity to be far greater than realized.

The above information was provided by the Speaker.

Objectives & Notes

Joaquim Fernandez Sola, M.D.

Date of talk: Thursday, June 8, 2006, 2:00pm

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Spain

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Training:

Current Job Description:	Medical Consultant in Internal Medicine
Current Faculty Appointments:	Chief of Chronic Fatigue Unit Hospital Clinic
Medical School/ University Attended	University of Barcelona, Fac Medicine
Internship:	Hospital Clinic, Barcelona, - Spain
Residency:	Hospital Clinic, Barcelona – Spain
Board Certifications:	Internal Medicine (Univ. Barcelona) Neurology (McGill Univ., Montreal)
Other Information: (including titles of books or articles you have recently written):	Book: Survive to Fatigue; Article: Chronic Fatigue Syndrome and Multiple Chemical Sensitivity After Insecticide Exposition

SPEECH TITLE: “Chronic Fatigue Syndrome Induced by Toxic Agents”

At the end of this Presentation, the participant should be able to:

1. Know diagnostic criteria for CHRONIC FATIGUE SYNDROME
2. Identify different precipitating agents of toxic origin in CHRONIC FATIGUE SYNDROME
3. Consider modifiable factors in toxic exposure leading to CHRONIC FATIGUE SYNDROME

The above information was provided by the Speaker.

CHRONIC FATIGUE SYNDROME INDUCED BY TOXIC AGENTS

Joaquim Fernandez-Solà M.D, Ph.D.

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AIM: This presentation considers the possible toxic origin of one of the most intriguing and also questioned diseases during the past two decades, the Chronic Fatigue Syndrome (CFS). One of the topics of the present "Symposium in man and his environment in health and disease" is Chronic Degenerative Disease, of which CFS is a clear example. Remarkably, the existence of this disorder has been questioned, its underlying pathophysiology debated, and an effective treatment opposed. The potential toxic origin of CFS has only recently been suspected.

BACKGROUND: CFS is the term characterized by the condition of persistent and unexplained fatigue resulting in severe impairment in daily functioning. Fatigue is a term that expresses failure to sustain force or power output. This is a subjective symptom experienced as a peripheral sensation, apparently located in muscular areas. However, its origin is usually central with failure in neurogenic transmission. The most widely scientifically supported is the CFS 1994 definition from the US Centers for Disease Control and Prevention, (Fukuda et al, 1994) which is now considered the standard. Case definition for CFS requires Fatigue for at least 6 months, as a new or definite onset, not being the result of an organic disease or of continued exertion, that is not alleviated by rest, and results in a substantial reduction in previous occupational, educational, social and personal activities. Concurrently, four or more other symptoms should be present for > 6 months such as impaired memory or concentration, sore throat, tender cervical or axillary lymph nodes, muscle pain, pain in several joints, new headaches, un-refreshing sleep, or malaise after exertion. Exclusion criteria consider medical conditions explaining fatigue, major depressive disorders, schizophrenia, dementia, anorexia nervosa, alcohol or substance abuse and morbid obesity.

The US community-based CFS studies found prevalence of 0.23 to 0.42 among adults, with higher rates in women being 75% of the cases. The mean age of onset is 29 to 35 years. Women are clearly more susceptible than men with a (ratio 6.5/1). Full recovery without treatment is rare. Marked persistent disability becomes established along the follow-up in at most 80% of CFS patients.

Many studies have investigated the *aetiology and pathogenesis* of CFS, being generally believed to be a multifactorial process. Non specific neurological, endocrine and immune system abnormalities have been described. Complex interactions between central regulation mechanisms are assumed to be at work. A model of disease with predisposing, precipitating and perpetuating factors is useful for understanding this complex disease. One of more factors in each category is conditioning but insufficient for the development of CFS. The predisposing factors which have been suggested to increase the risk of CFS in adults are genetics, personality and lifestyle, inactivity in childhood, and inactivity after infectious diseases. Among the precipitating factors acute physical or psychological stress might trigger the onset of CFS. Infections such as cold, flu-like illness or infectious mononucleosis may precipitate the onset in three quarter of cases. Toxics have not been considered seriously as precipitating factors until only recently. Perpetuating factors such as a strong belief in a physical cause of the illness, a strong focus on bodily sensations, and a poor sense of control over complaints contribute to an increase in fatigue.

Another characteristic of CFS is the frequent existence of comorbidity such as Fibromyalgia, multiple chemical sensitivity, sicca syndrome or irritable bowel syndrome. In our experience, patients with CFS share a mean of 4.2 other related diseases, being Fibromyalgia the most frequent (up to 70% of cases). The presence of comorbidity increases the severity of CFS.

TOXIC CHEMICAL AGENTS are potential precipitating factors in CFS. However, review of the literature only shows isolated data in this field. Agriculture, industrial areas and accidental situations are potentially involved. Ingestion of ciguatera in tropical countries, and inhalatory exposure to carbon monoxide, solvents, hydrocarbons, trichloroethylene, sulphydric acid and specially organophosphate (OP) pesticides are the most frequent toxics triggering CFS. Clearly, OP are the most frequently involved toxic agents in CFS. Although chronic neurotoxic effects of OP have been previously described, with the term OPICN (organophosphorous-ester induced chronic neurotoxicity), the authors did not specifically identify these symptoms as CFS. Probably the neurocognitive impairment that characterizes CFS may be identifiable with the reported OPICN situation after OP exposure.

PROSPECTIVE STUDY: (Fernandez Sola J, Lluís M, Nogue S, Munne P. *Chronic Fatigue Syndrome and Multiple Chemical Hypersensitivity after insecticide exposition. Med Clin (Bar) 2005; 124:451-453*)

Method: In Hospital Clinic, a major university teaching-hospital downtown Barcelona, Spain, we have frequently observed patients that, after exposure to insecticide, have developed a diversity of symptoms, in which physical and mental fatigue was prevalent. It was supposed that OP exposure could be a precipitating factor of CFS. Therefore, we embarked upon a study to evaluate the relationship between CFS and toxic insecticide exposure. Along a 4-year

period, 39 patients who attended the Toxicology Unit of the Hospital Clinic after insecticide exposure were evaluated. The circumstances of plaguicide exposure, such as in the workplace or accidental, the type of insecticide, the method of application and the period comprised between fumigation and patient contact were reported. Clinical evaluation comprised assessment of symptoms including acute mucosa irritation, muscarinic or nicotinic manifestations, neurocognitive impairment, gastrointestinal manifestations and muscle pain related to fibromyalgia. In addition, patients reporting physical or mental fatigue of more than 6 months were referred to the Chronic Fatigue Unit and studied to validate CFS diagnostic criteria (Fukuda, CDC, 1994). Quality of life (SF-36), Daily functional impairment (H.A.Q) were evaluated by specific questionnaires and the functional degree of fatigue was determined with a progressive scale (grades I-IV).

Results: From an initial group of 35 patients exposed to insecticides, we included 29 patients, who fulfilled criteria of CFS in the study. Twenty-two were women with a mean age of 45 ± 8 years. We observed that 96% developed a chronic neurocognitive syndrome with impairment of short-term memory and concentration; 85% developed acute mucous irritative syndrome, 50% fibromyalgia, and 27% gastrointestinal manifestations. No patient showed manifestations of acute intoxication, and all showed normal levels of blood cholinesterase.

The type of plaguicide was organophosphate in 9 cases, pyrethrin in 9 cases and mixed OP and pyrethroid in 8 cases. The circumstances of exposure were in the workplace in 26 cases and accidental in 3. Hotels, administrative public offices, commercial and health primary centres were the most frequent workplaces of exposure to insecticides in this series. In 81% of cases, there was an outbreak situation, with a mean of 4.2 cases each. Time between fumigation to patient exposure was lower than 24 hours in 11 cases (42%). This is the legal safety period after fumigation in Spain.

The severity of fatigue was slight (grade 1) in 65% of cases and moderate (grade 2) in 35%.

During the follow-up, along a mean period of 2.5 ± 0.9 years, 23% of patients had permanent working disability, 56% a transitory working disability, and 19% had a self-limited episode of less than one year in duration. This percentage of permanent disability increased over time.

In conclusion, we observed precipitation of Chronic Fatigue Syndrome in two-thirds of the patients attended to a Toxicology ward after organophosphate exposure. In most of the cases, prevention and safety measures had not been adequately accomplished. Consequently, we suggest that fulfillment of fumigation safety rules in areas with human contact, mainly in workplaces, is necessary.

DISCUSSION. Pathogenesis of pesticide-induced CFS is not completely known. It has been suggested that OP and pyrethrin exposure increases systemic cholinergic activity. This is due to a directly OP induced decrease in esterase activity at a vascular, endothelial and cerebral level. Disturbances in brain GABA activity and increased oxid nitrate activity have also been proposed, which lead to a Neuronal Hyperexcitability that is the basis of the induction of CFS.

During the 18-year experience in our CFS Unit, around 15% of patients who consult for chronic disabling fatigue have been previously exposed to potential toxic agents that may trigger CFS. OP and pyrethrin insecticides hydrocarbons and industrial solvents are the most frequent agents, with the exposure usually being of working origin. Although toxic exposure should be chronic and repetitive at low-dose, occasional acute high-dose exposure may also be enough to trigger CFS. In addition, the incidence of toxic agents as precipitating factors of CFS seem to increase over time.

Since CFS is a progressive disabling disease, with increased incidence and without effective treatment after its establishment, specific prevention measures are necessary to avoid this 15% percentage of toxic-induced development of this disease. In the future, toxic-induced CFS may increase even more if specific interventions are not established.

Objectives & Notes

Richard Jaeckle, M.D.

Date of talk: Thursday, June 8, 2006, 2:30pm

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Training:

Current Job Description:	Private Practice of Psychiatry and Environmental Medicine
Medical School/ University Attended	University of Texas Southwestern Medical School
Internship:	Veterans Administration Hospital, Dallas, TX
Residency:	Psychiatry: St Louis Univ Hospitals & Child Psychiatry: Washington Univ Child Guidance Clinic
Board Certifications:	AmerBdPsyNeurol:Psychiatry; AmerBdPsyNeurol: Child Psychiatry; AmerBdEnvironMed

SPEECH TITLE: "TAAT* Support in Chronic Illness"
***Targeted Amino Acid Therapy**

At the end of this Presentation, the participant should be able to:

1. To relate the connection between the stress of chronic illness and neurotransmitters
2. To elaborate the role of TAAT in the normalization of the stress reaction and neurotransmitters
3. To explain the method of urine collection and the TAAT protocol

The above information was provided by the Speaker.

TAAT* Support in Chronic Illness

*Targeted Amino Acid Therapy

Richard G. Jaeckle, M.D.
Professional Association
FAACAP, FAAOA, FAAEM

The brain' reaction to chronic illness and any other kind of stress is the activation of neurotransmitter release. Such activation can lead to depletion of neurotransmitter reserves and the compounding or spreading of symptoms to include mental symptoms of behavior, mood or cognition. Restoration of reserves and the reduction of secondary stress of mental symptoms is the goal of TAAT.

The testing of neurotransmitters via a morning urine specimen will be presented. The phased treatment with neurotransmitter precursors will be demonstrated. Progress in the normalization of urinary neurotransmitter levels and symptoms is shown.

Objectives & Notes

Allan D. Lieberman, M.D.

Date of talk: Thursday, June 8, 2006, 3:30pm

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Training:

Current Job Description:	Medical Director of Center for Occupational and Environmental Medicine
Medical School/ University Attended	Chicago Medical School
Internship:	Mt. Saini Hospital – Chicago
Residency:	Children’s Memorial Hospital – Chicago
Board Certifications:	American Board Env. Medicine

SPEECH TITLE: “Hydraulic Fluid Toxicity in a Surgical Suite”

At the end of this Presentation, the participant should be able to:

1. Understand epidemiology of Hydraulic Fluid Toxicity
2. Understand Toxicology of Hydraulic Fluid
3. Understand the Treatment of Management of Hydraulic Fluid Toxicity

The above information was provided by the Speaker.

HYDRAULIC FLUID CONTAMINATION IN A SURGICAL SUITE

Allan D. Lieberman, M.D.
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During the months of November and December of 2004, approximately 3800 patients in Durham and Raleigh North Carolina were exposed to surgical instruments unknowingly contaminated with waste hydraulic fluid inadvertently stored in containers labeled as surgical instrument detergent. It has not been determined how many patients developed complications of their health as a result of exposure. But there are at least 30-50 who have made themselves known to a support group of injured patients.

A compilation of signs and symptoms was found from this group. See Table 1.

Table 1. Signs and Symptoms of Patients Exposed to Hydraulic Fluid

- extreme fatigue
- headaches
- dizziness
- memory difficulties
- behavioral changes, especially irritability
- swelling or puffiness at or near incision site, even months later
- wounds that oozed liquid (clear, yellow, or green) for months, but doctors said there was no infection
- continued pain at the surgical site, mild or severe
- delayed healing of joints
- joint pain, even in areas not near the surgical site, beginning only after surgery
- wounds that took a very long time to heal
- unexplained rashes
- hair falling out
- swollen lymph nodes
- weight loss
- gastrointestinal pain or upset
- nausea / vomiting

Case Presentation 1: Allan Lieberman, M.D.

Case Presentation 2: William Rea, M.D.

Discussion: **Toxicology of Hydraulic Fluids**

More than 200 million gallons of hydraulic fluids are sold each year in the United States according to a government document published in 1997. Hydraulic fluids can be divided into seven chemical classes:

- phosphate esters,
- mineral oil-in-water and,
- water-in-oil fluids,
- polyalphaolefin oligomers,
- polyhalohydrocarbons,
- polyglycols,
- silicate esters, and
- silicones.

Mineral-oil based hydraulic fluids (including fire-resistant mixtures of mineral oil and water) have been estimated to comprise 98% of the world demand for hydraulic fluids (1980 reference). These fluids are made from dewaxed petroleum-based crude oils that are blended with additives such as corrosion inhibitors (e.g. fatty acids), oxidation inhibitors (e.g., phenols, amines, and sulfides), defoamers (e.g silicone oils), and antiwear additives (e.g. organophosphates). Thus, mineral-oil-based hydraulic fluids are complex mixtures of aliphatic and aromatic hydrocarbons to which other compounds have been added.

Common types of organophosphate esters include:

- tricresyl phosphates,
- tributyl phosphates, and
- tertiary butylated phenyl phosphates.

Organophosphate ester hydraulic fluids and additives often contain mixtures of organophosphate esters.

Hydraulic fluids themselves cannot be measured in blood, urine, or feces, but certain chemicals in them can be measured. Aliphatic hydrocarbons, which are major components of mineral oil hydraulic fluids and polyalphaolefin hydraulic, can be detected in the feces.

Most experience from hydraulic fluid toxicity comes from the aircraft industry with 760 incidents involving 900 flight attendants subsequent to 1989. The symptoms associated with these flight attendants included:

- fatigue / loss of energy
- headaches
- blurred vision
- nausea / vomiting
- dizziness
- swollen lymph nodes
- confusion
- giddiness
- disorientation
- lack of motor control
- loss of cognitive function
- tremors

What is unique about our two cases and the 30 – 50 others is the portal of entry into the body – skin, mucosa, soft tissue, and visceral contact.

Mechanism of Injury

1. Chemicals can injure directly by their irritant inflammatory effect on tissue as well as by foreign body response in the tissue. This could account for the breakdown of surgical wounds and delay in healing.
2. But they can have specific effects:
 - Neurotoxic
 - Ototoxic
 - Immunotoxic
 - Metabolic toxicity

Autoimmunity is a major consequence of toxic chemical exposure. This mechanism of injury could explain the chronic nature of these exposed patients.

Another immunologic mechanism that can explain the recurrent infections in these patients is the modulation of IL-2 driven proliferation of lymphocytes by anticholinesterase organophosphate additives to the hydraulic fluids. (See Bavari et al.)

Proposed Treatment

Biodetoxification – is the only reasonable approach to reduce the body burden of hydraulic fluid constituents in their bodies.

Medical Literature References

Sverdrup B, Kallberg H, Bengtsson C, Lundberg I, Padyukov L, Alfredsson L, Klareskog L; Epidemiological Investigation of Rheumatoid Arthritis Study Group.

Association between occupational exposure to mineral oil and rheumatoid arthritis: results from the Swedish EIRA case-control study. *Arthritis Res Ther.* 2005;7(6):R1296-303. Epub 2005 Sep 23.

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The arthritogenic adjuvant squalene does not accumulate in joints, but gives rise to pathogenic cells in both draining and non-draining lymph nodes. *Clin Exp Immunol.* 2002 Mar;127(3):430-5.
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- Witkowski C. Remarks on airliner air quality ASHRAE Conference. Chicago IL. Jan 24, 1999.

24th ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT Schedule

Friday, June 9, 2006

8:00 **ANNOUNCEMENTS/MODERATOR:** Larry Plumlee, M.D.

8:05 George Yu, M.D., "Toxic Load in Fat Compartments Especially the Visceral Fat"

8:25 Q & A

8:35 Sherry Rogers, M.D., "Detoxification for Reversing End-Stage Chronic Diseases, Part I"

8:55 Q & A

9:05 Jerry Alter, Ph.D., "Scavenging Enzymes and Chemical Sensitivity"

9:25 Q & A

9:35 Doris J. Rapp, M.D., "Genetic Engineered Food"

9:55 Q & A

10:05 BREAK WITH EXHIBITORS

10:50 Jean Monro, M.D., "Man's Sense of Awareness Illustrated by Visual Processing"

11:10 Q & A

11:20 Klaus-Dietrich Runow, M.D., "Poisoned Children - Toxic Metal Intoxication in Kosovo"

11:40 Q & A

12:00 OPEN LUNCH

1:30 **MODERATOR:** Jean Monro, M.D.

1:30 Hiroaki Kumano, M.D., Ph.D., "The Impact of Stress-Related Factors on MCS"

1:50 Q & A

2:00 Kenneth Fine, M.D., "Treating Systemic Inflammation from an Intestinal Viewpoint"

2:20 Q & A

2:30 Sherry Rogers, M.D., "Detoxification for Reversing End-Stage Chronic Diseases, Part II"

2:50 Q & A

3:00 BREAK WITH EXHIBITORS

3:45 Mohamed B. Abou-Donia, Ph.D., "Involvement of Tri-*ortho*-Cresyl Phosphate in Aircrew Symptoms"

4:05 Q & A

4:15 Panel Discussion/Case Studies: William J. Rea, M.D. and Allan D. Lieberman, M.D.

6:00 RECEPTION IN EXHIBIT ROOM

FRIDAY, JUNE 9, 2006

ABSTRACTS

AND

HANDOUTS

Objectives & Notes

George Yu, M.D.

Date of talk: Friday, June 9, 2006, 8:05am

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Training:

Current Job Description:	Surgeon
Current Faculty Appointments:	Clinical Professor of Urology George Washington University
Medical School/ University Attended	Tufts University 1973
Internship:	University of Pennsylvania
Residency:	General Surgery – Harvard – Peter Bent Brigham Urology-specialty -Johns Hopkins Medical Center
Board Certifications:	American Board of Urology - 1983
Other Information: (including titles of books or articles you have recently written):	1) Text book-1996 Critical Operative Maneuvers in Urologic Surgery – Mosby; 2) Reviewer for Journal “Urology;” 3) NIH review for CAM nutritional intervention for cancer; 4) Consultant to FASC – 911 Firefighter detoxification.

SPEECH TITLE: “Toxins in Visceral Fat and Correlation to other Fat Compartments of the Body”

At the end of this Presentation, the participant should be able to:

1. To establish a relative coefficient ratio of toxic load between visceral fat and other fat compartments of the body.
2. To illustrate a simple technique for fat aspiration using “Coleman” needles

The above information was provided by the Speaker.

Toxins in Visceral Fat and Correlation to other Fat Compartments of the Body

George Yu, M.D. and John Laseter, Ph.D.

It is well known that most toxins are lipophilic and therefore accumulates in the fatty adipose tissues and lipid rich organs (brain) compared to the serum blood compartment of the body.

Yet there has been a trend of decreasing fatty tissue sampling for toxins as the procedure may carry higher morbidity to subjects, inconveniences to investigators, and more extensive bureaucratic barriers for study protocols.

Historically, review of the literature shows that much of the fatty tissue sampling for toxins have been obtained from random autopsies, and random surgical fat sampling of subjects in regions of toxin prevalence. In contrast, exact measurements should be done on subjects in regions of toxin prevalence. In contrast, exact measurements should be done on subjects truly exposed to specific toxins and comparisons of their fatty tissues to blood toxins levels are then more meaningful and valid. True studies show comparative fatty tissues to be 100-200x the level of blood serum. The measurements should be done as nanograms per milliliter; as a volume to volume denominator comparison.

We have sampled from 6 surgical patients with renal cell carcinomas, renal benign mass and prostate cancer and measured toxin levels in multiple fat compartments and tumor itself. Levels of PCB ranged in between 60x to 100x higher than blood, and levels of chlorinated pesticides of DDE ranged between 300x to 200x higher than blood.

Our goal was to establish a correlation coefficient of toxin levels in the various fatty tissue compartments of the body. We measured peripheral fat, visceral fat in the form of mesenteric fat, falciform ligaments of the liver, omentum, retroperitoneal fat, Gerot's fat around the kidney, and the kidney and prostate organ.

Visceral fat has gained wide scientific and medical interest in the last few years and we have discovered its association with cardiovascular diseases, cancer diseases, as well as metabolic and endocrine diseases such as hyperinsulinemia and diabetes.

We have also discovered Leptin and two other new hormones within fat. One cannot help but consider the potential importance of toxins in visceral fat adjacent to functioning organs on long-term pathogenesis of disease.

An incidental observation was noted at the National Institute of Health on February 25, 2002, when a panel of 15 scientists reviewed cases of nutritional interventions in patients with terminal cancer diseases under the "Best Case Series" program. The review showed an unexpected finding of decreasing visceral fat as the cancer regressed by CAT Scan analysis. Thus the term of "Visceral Defatting" came into existence. Is there a relationship between fat decompression and detoxification and carcinomatosis?

The Daum procedure has been the standard of fatty tissue biopsy in the past. However, with advances in plastic surgery, we can now use the newer technology to simplify fat extraction for toxin analysis with least patient morbidity. Using the modern "Coleman" reusable needles for fat extraction, we can obtain 5 to 50 milliliters of fat without difficulty. Instead of using the fat around the buttocks which is uncomfortable for subjects when sitting, we can obtain fat from the periumbilical area.

As strategies for detoxifications are being refined, we should be able to use fat as well as blood for assessment of the efficacy of treatments with pre and post analysis. The importance of establishing some form of correlation coefficients of peripheral fat to other fat compartments such as the visceral fat will become of clinical importance as well as providing further understanding of toxins long-term effects on the viscera leading to disease.

Objectives & Notes

Sherry Rogers, M.D.

Date of talk: Friday, June 9, 2006, 8:35am

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Training:

Current Job Description:	Physician/Author
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Internship:	S.U.N.Y. Health Sciences Center at Syracuse
Board Certifications:	Family practice and Environmental Medicine
Other Information:	Detoxify or Die, The High Blood Pressure Hoax, No More Heartburn, Pain Free in 6 Weeks, Tired or Toxic, The Cure is in the Kitchen, Wellness Against All Odds, Monthly referenced newsletter Total Wellness and much more.

SPEECH TITLE: **“Detoxification for Reversing End-Stage Chronic Diseases, Part I”**

At the end of this Presentation, the participant should be able to:

1. Using hypertension as an example, to appreciate that diuretics, the first line treatment directive for disease “management”, lower magnesium, potassium, CoQ10 and raise homocysteine, fostering the need for more medications
2. Know that there are many simple and inexpensive cures for hypertension, depending on the biochemistry of the individual
3. Know that phthalates are ubiquitously unavoidable human xenobiotics (average daily intake 3 gm) that can mimic or serve as the backbone for any chronic disease, including hypertension, and stall healing indefinitely (see Part II for their diagnosis and treatment).

Disclosures:

Author of over a dozen highly referenced books and monthly newsletter on healing of chronic “incurable” diseases, and has received small honorarium from Carlson Laboratories and MetaMetrix Laboratories in the last year for non-CME presentations

The above information was provided by the Speaker.

Detoxification for Reversing End-Stage Chronic Diseases, Part I

Sherry A. Rogers, MD, ABFP, ABEM, FACAAI, FACN

Abstract:

No longer do we have to be content with the management of diseases with pharmaceuticals as practice guidelines instruct us, for we can cure them. We can actually reverse the pathophysiology and not just shift symptoms to other vulnerable target organs. Over 2000 references substantiate molecular biochemical mechanisms of what has been done in clinical practice for over three decades to make diseases disappear.

Finding the true underlying biochemical causes of disease are ignored, in favor of biochemically poisoning the symptom-inducing pathway with a pharmaceutical specifically designed to inhibit life processes. Using hypertension as an example, practice guidelines dictate diuretics as the first line treatment for lifelong management of the disease. But enormous literature shows how diuretics deplete not only potassium, but magnesium. And either of these could be the underlying correctable cause of the hypertension. Yet it doesn't stop there, for they also lower CoQ10 and raise homocysteine 16%. And this does not cover all the undesirable side effects of this one category of drugs, much less all the others that are commonly prescribed for hypertension. Clearly once started on a drug, the need for further drugs for escalating symptoms as well as new symptoms snowballs out of control as poisoning pathways allows underlying nutrient deficiencies to remain ignored while further depleting nutrients.

Besides nutrient deficiencies as the underlying causes of chronic diseases, elevated xenobiotics is another high-ranking cause, using phthalates here as an example. What started out naively as the moniker "environmental endocrine disruptors" has blossomed into what they really are, **total metabolic disruptors**. Targeted molecular biochemistry assays highlight necessary nutrient corrections that allow membrane repair and detoxification to restore function to insulin receptors, cytokine release sites, neurotransmitter ports, intracellular matrix communication and reactivate ion channels. And unloading PPAR (peroxisome proliferator-activated receptors), paralyzed by phthalates (dietary, medicinal and environmental plasticizers), as an example, restores beta-oxidation for fatty acid synthesis, detoxification and healing. Part II will explore how this is done.

Conclusion: Drugs merely poison malfunctioning pathways, temporarily reducing symptoms but leading to new ones as drugs further deplete detoxification nutrients and foster ignoring repair of causative defects. The beauty of a program to identify biochemical defects is that this corrects the underlying biochemical abnormalities (sometimes dating back to prenatally as recent government studies show) and unloads xenobiotics sufficiently to allow activation of natural healing mechanisms. This program is significantly less expensive, less toxic, and heals versus suppressing symptoms with lifelong expensive drugs, enabling chronic diseases to virtually disappear. It is done at home with non-prescription items under physician guidance, which is the purpose of this presentation. We have the tools for a new generation of physicians trained in not merely "managing" disease, as practice guidelines instruct, but actually curing diseases.

References:

Alonso-Magdalena P, et al, The estrogenic effect of bisphenol A disrupts pancreatic B-cell function in vivo and induces insulin resistance, *Environ Health Perspect* 114:106-12, 2006

Rogers SA, *The High Blood Pressure Hoax*, 2005 (for over 700 more references), prestigepublishing.com

Lombardo YB, et al, Effects of dietary polyunsaturated n-3 fatty acids on dyslipidemia and insulin resistance in rodents and humans, A review, *J Nutr Biochem*, 17:1-13, 2006

Narayanan BA, et al, Docosahexaenoic acid regulated genes and transcription factors inducing apoptosis in human colon cancer cells, *Int J Oncol*, 19:1255-62, 2001

Objectives & Notes

Gerald Alter, Ph.D.

Date of talk: Friday, June 9, 2006, 9:05am

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Training:

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Medical School/ University Attended Internship:	Washington State University, Ph.D.; Albion College, BA Postdoc – Harvard Medical School

SPEECH TITLE: “Scavenging Enzymes and Chemical Sensitivity”

At the end of this Presentation, the participant should be able to:

1. evaluate potential organophosphate and formaldehyde chemical sensitivity based on scavenging enzyme hypothesis for the genesis of chemical sensitivity
2. correlate abnormal activities of several blood enzyme activities with formaldehyde and organophosphate chemical sensitivity
3. identify correlations between chemical sensitivity and vitamin and nutritional deficiencies

The above information was provided by the Speaker.

Abstract: Scavenging Enzymes and Chemical Sensitivity
Gerald M. Alter, Ph.D.

We have hypothesized that Multiple Chemical Sensitivity (MCS) in humans is linked to abnormal levels of enzymes capable of metabolizing respective toxins to less toxic products. To test this assertion with respect to specific sensitivities toward organophosphates (OP) and formaldehyde (FORM), we have examined the levels of several enzyme activities in several clinically accessible tissues. Enzymes activities used in these studies include: aldehyde dehydrogenase (ALDH), chi alcohol dehydrogenase (χ -ADH), paraoxonase (PON), and aryl esterase (AE). Results of previous studies demonstrate that blood of normal individuals has both sufficient activities of these enzymes and is sufficiently available to be useful in our analyses. Blood samples collected from individuals in this investigation were separated further into fractions enriched in RBCs, WBCs, and serum. Activities were routinely determined for each of these fractions. In current studies, blood samples from populations of OP sensitive (only), chemically sensitive including OP sensitive, FOR sensitive (only) and chemically sensitive including FOR sensitive were analyzed, and results compared with blood samples from a group of normal individuals. Experimental groups were recruited from patients clinically tested for specific chemical sensitivity.

Our results indicate that several of the enzyme activities in several of the blood preparations are lower in chemically sensitive individuals than in the control population. Segregation of chemically sensitive individuals from normal individuals was best when pairs of activities were considered. The activity pair, aryl esterase from the red cell blood fraction and aldehyde dehydrogenase for the white cell blood fraction, was best in distinguishing OP sensitive from normal individuals. Though the segregation was only significant at the $p=0.06-0.08$ level when comparing OP sensitive (only) with normal individuals, the segregation was significant at the $p=0.005-0.01$ level when normal and chemically sensitive including OP sensitive groups were compared. The activity pair PON from the red cell fraction and aldehyde dehydrogenase from the white cell blood fraction was best in distinguishing FOR (only) sensitive form normal individuals. When chemically sensitive (including FOR) were compared with normals, the statistical significance of the distinction did not improve. By increasing the sample size, particularly the FOR only and the OP only groups, we anticipate that statistical significance of these results will significantly increase. Group sizes were small (9 and 10 for uniquely sensitive individuals, 21 and 28 with multiple chemical sensitivities, and 20 individuals in a control groups.) We are currently addressing this issue. However, results at this point suggest a novel link between the phenotypic expression of chemical sensitivity and the biochemical markers of enzyme activity in blood that can be quantitatively measured. This information may be clinically useful to help identify or confirm a diagnosis of chemical sensitivity and suggest novel approaches in managing this malady.

Objectives & Notes

Doris Rapp, M.D.

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Training:

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Current Faculty Appointments:	Emeritus University Appointment/Clinical Professor of Pediatrics at SUNYAB
Medical School/ University Attended	New York University/University of Buffalo
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Residency:	Children's Hospital of Buffalo/Buffalo General Hospital
Board Certifications:	Pediatrics, Pediatric Allergy and Environmental Medicine
Other Information: (including titles of books or articles you have recently written):	Best selling author of "Is This Your Child." Latest book "Our Toxic World – A Wake up Call"

SPEECH TITLE: "Genetic Engineering"

At the end of this Presentation, the participant should be able to:

1. Know what GE is and how extensive it has become.
2. Understand GE pros and cons
3. Have more appreciation of its harmful effects on our food supply, farmers and economy.

The above information was provided by the Speaker.

Genetic Engineering

Doris J. Rapp, M.D.
Scottsdale, AZ

Genetic engineering is progressively more evident in American food, plant, animal and pharmaceutical products. Genetic engineering creates variations in normal gene patterns and can damage an organism's DNA. GE can make products grow bigger and faster, prevent some plant or animal disease or protect or reduce some product's response to herbicides. This entire process is, however, challenging to control. Even under the most stringent laboratory conditions it can lead to the formation of allergens, toxins and diminished nutritive value within a substance. The following will be discussed:

What is genetic engineering? How is it done?

What are the pros and cons of GE?

How extensive is GE; which products are affected?

How do growth hormones and GE foods affect our food supply?

What do some renowned scientists say about GE?

How many studies show GE safety in children or adults?

Why should our EPA, government or other governments be concerned?

What does GE do to farmers, the ecosystem, agribusinesses and our economy?

Conclusion: GE is insufficiently studied and potentially can be exceedingly dangerous. The safety of GE products has not been properly or completely evaluated. Many foreign countries are increasingly skeptical about the safety of GE products. Animal studies provide disturbing evidence of profound undesirable health effects due to GE. Human research is woefully insufficient but the suppression of some undesirable GE findings in rats by Dr. Arpad Pusztai in Scotland is most alarming. (www.projectcensored.org/storirs/2001/7.html)

References: Article: **Assessing the Safety and Nutritional Quality of Genetically Engineered Foods**, A Science-based, Precautionary Approach.

Book: **Genetic Engineering, The Hazards, Vedic Engineering, The Solutions**, John Fagin (Amazon.com), John Fagan, PhD 515 472 8341

Objectives & Notes

Jean Monro, M.D.

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Other Information:	Treatment of cancer with mushroom products. Arch environ Health 2003; 58:533-7

SPEECH TITLE: “Man’s Sense of Awareness Illustrated by Visual Processing”

At the end of this Presentation, the participant should be able to:

1. Recognize man’s sense of awareness, that his sixth sense is real, and is impinged upon by frequencies, including neutralizing vaccines and light, and can be modulated.

The above information was provided by the Speaker.

Breakspear Hospital



Title: **MAN'S SENSE OF AWARENESS ILLUSTRATED BY VISUAL PROCESSING**

Venue: **24TH ANNUAL INTERNATIONAL SYMPOSIUM ON MAN AND HIS ENVIRONMENT,
DALLAS, 9TH JUNE 2006**

Author: **JEAN A MONRO
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Medical Director, Breakspear Hospital, Wood Lane, Hemel Hempstead,
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In Association
with: **IAN JORDAN, FBDO CL**

A B S T R A C T

Our work has shown that we can trigger responses in people using neutralising vaccines, which can be applied cutaneously or intradermally. We have demonstrated that these methods work through their effects in transmitting information about frequencies through the neural pathway for allergy.

This talk and film demonstrate that using frequencies through the entire colour spectrum is another way of achieving the same type of control as with the use of neutralising vaccines, in that responses to different shades of colour represent a range of frequencies, which can either provoke symptoms, or nullify them. Control of symptoms can be achieved with colour integration through the visual pathway, or independent of the visual pathway, through the skin. Man's sense of awareness represents a perception of particles and frequencies, and is global through the body as a whole.

Our film has shown the integrative visual pathway effects on other systems.

Jean A Monro

Medical Director

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Objectives & Notes

Klaus-Dietrich Runow, M.D.

Date of talk: Friday, June 9, 2006, 11:20am

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Training:

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Board Certifications:	Internat. Board of Environmental Medicine (USA)
Other Information:	Books: Klinische Oekologie (Clinical Ecology) and Nervenschutz durch Entgiftung (Nerve Protection by Detoxification). First honorary member of the German Society for Environmental and Human Toxicology; The society for Threatened Peoples of Goettingen, Germany, brought me to Kosovo to test for toxic heavy metals in refugee camps near Mitrovica

SPEECH TITLE: “Poisoned Children – Toxic Metal Intoxication in Kosovo”

At the end of this Presentation, the participant should be able to:

1. Estimate the toxic load from a group of more than 500 Roma people living at the edge of the derelict Trepca mines near Mitrovica / North Kosovo .with the slag heaps that waft clouds of heavy metal containing dust into the air, water and soil.
2. See which kind of symptoms and diseases developed the IDP (internally displaced people) during the last 6 years.

The above information was provided by the Speaker.

Poisoned Children – Toxic Metal Intoxication in Kosovo

Klaus-Dietrich Runow

Speech #1 Friday, June 9, 2006, 11:20am

More than 14,000 Romani homes were looted and destroyed after KFOR troops arrived in 1999. Only a few hundred of those homes have been rebuilt. No one has been allowed to go back to the job they had before the war. Since they were burned out of their homes during the Kosovo war in 1999 a group of more than 500 Roma people living at the edge of the derelict Trepca mines near Mitrovica / North Kosovo. The toxic load comes from the slag heaps that waft clouds of heavy metal containing dust into the air, water and soil.

On 19th October 2005, the Society for Threatened Peoples of Goettingen, Germany, brought me to Kosovo to test for toxic heavy metals in the IDP (internally displaced people) camps near Mitrovica: Zitkovic and Chesmi Lug. Hair samples were collected from 64 persons (49 children between the ages of 1-15) and 15 adults. Analytical method: High Resolution ICP-MS (Inductively Coupled Plasma Mass Spectrometry). In addition to Lead we determined other heavy metals, including Aluminum, Antimony, Arsenic, Cadmium, Manganese, Tin etc. and we measured minerals and trace elements – together 39 elements.

The analysis was completed between 28th and 31st October 2005. Results: All analysis showed extreme high levels of Antimony, Arsenic, Lead, Cadmium and Manganese. In only one sample the Arsenic level was in the reference range. 62 hair samples (97%) showed high Aluminum levels, 44 hair samples (69%) high Vanadium levels und 37 hair samples (58%) high Mercury levels.

Regarding Lead the readings range from 20 to 1200 µg/g (Reference Range < 1,0 µg/g). The analysis showed disturbingly low levels of Selenium, which is essential for thyroid and heart function, immune system and for binding and inactivating toxic heavy metals.

Selenium deficiency is associated with cardiovascular disorders. Symptoms from low levels of selenium include arthritis, heart diseases (dilatative cardiomyopathy), hurting muscles, muscle weakness, losing weight, losing hair, changing of hair structure, suppression of the immune system, reduction of fertility and eye diseases. Pediatricians see a connection between low Selenium levels and SIDS (Sudden Infant Death Syndrome).

More specifically the lead levels from the Roma children were:

- 8 Children had readings between 20 - 100 µg/g
- 9 Children had readings between 101-200 µg/g
- 13 Children had readings between 201-300 µg/g
- 4 Children had readings between 301-400 µg/g
- 2 Children had readings between 401-500 µg/g
- 4 Children had readings between 501-600 µg/g
- 3 Children had readings between 601-700 µg/g
- 6 Children had readings between 701-1200 µg/g

Reference Range of Lead in hair < 1,0 µg/g

Such high lead level readings are unprecedented in the world and pose an extreme health risk to the children in the camps. Adults are at risk as well especially pregnant women. In 2004 WHO conducted blood tests on several children in the camps after a four-year-old Romani girl died of lead poisoning. All children tested had dangerously high lead levels. WHO recommended immediate evacuation but the people still live in the contaminated area.

In 2005 Kosovo Roma Refugee Foundation followed the pregnancies of 50 Romani women in the three camps. Only six children were born, all suffering from mental retardation. Four other children were still-born. The rest of the pregnancies ended in miscarriage. Medical doctors familiar with the camps and the conditions attributed the miscarriages, still-births and mental retardation to the lead poisoning.

Medical treatment for the first patients out of the refugee camp started in April 2006 in our clinic in Bad Emstal/Germany.

First observations and results regarding the effectiveness of the detoxification therapy will be presented in Speech # 2.

Klaus Dietrich-Runow, Institute for Functional and Environmental Medicine (IFU)
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Special thanks to the Society for Threatened People (GfbV), Goettingen,Germany, www.gfbv.de : Tilman Zülch, Jasna Causevic, Frank Witte and Paul Polansky who is living in Kosovo and has been struggling since over 6 years for the evacuation of the polluted camps.

Objectives & Notes

Hiroaki Kumano, M.D., Ph.D.

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Medical School/ University Attended	The University of Tokyo
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Board Certifications:	Certified Doctor for Psychosomatic Medicine
Other Information: (including titles of books or articles you have recently written):	Symptom profile of multiple chemical sensitivity in actual life. Psychosom Med 67:113-124, 2005; Use of QEESI questionnaire for a screening study in Japan. Toxicol Ind Health 21:113-124, 2005; Application of Quick Environment Exposure Sensitivity Inventory (QEESI) for Japanese population: study of reliability and validity of the questionnaire. Toxicol Ind Health 19:41-49, 2003

SPEECH TITLE: “**The Impact of Stress-Related Factors on MCS**”

At the end of this Presentation, the participant should be able to:

1. Know the role of psychosocial stressors on the onset and progression of MCS.
2. Know what kinds of psychosocial individual vulnerabilities MCS subjects have.
3. Know what kinds of symptoms MCS subjects complain of.

The above information was provided by the Speaker.

The impact of stress-related factors on multiple chemical sensitivity ¹⁾

Hiroaki Kumano

Department of Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo

Goals and objectives: The objective of this study was to clarify the impact of stress-related factors on the onset and progression of the multiple chemical sensitivities (MCS). Our hypotheses of this study were the following: 1) Relevant psychosocial stressors as well as chemical exposure can be identified before the onset of MCS; 2) Psychosocial individual differences such as personality, stress coping, social support, and life style profile are present related to stress vulnerability; 3) The patients have multiple signs and symptoms including somatic and psychological symptoms, psychiatric comorbidity, and autonomic dysfunction.

Methods: A cross-sectional comparison was made of the following groups: 1) Patients who was diagnosed multiple chemical sensitivities in the Clinical Environmental Center of the Kitasato Institute Hospital (N = 27); 2) Normal controls who was recruited through magazine advertising and had not developed MCS in spite of moving to new houses or reforming their houses during the past three years (N = 33). All subjects answered the battery of psychosocial questionnaires, underwent the structured interviews for diagnosing psychiatric comorbidity, and were assessed on their autonomic nervous function based on heart rate variabilities either in the examination room in the hospital or in their own houses. The score of life events during the past one year was higher in patients than in controls although its difference did not reach significant level ($p=0.079$). There were no significant differences in various personality measures, stress coping style and social support between MCS and control groups. Although remarkable features were noted in some life style measures such as no smoking habit and little alcohol drinking in the past one month in MCS group, there were no differences of those habits between the two groups before the onset of MCS assessed by retrospective questions. MCS patients had many physical symptoms compatible with its diagnostic criteria, and the psychiatric comorbidity was as high as 89 % in MCS group compared to 11 % in control group ²⁾. However, the patients did not complain of as many psychological symptoms as expected, and the measures of heart rate variability's were not different between the two groups.

Conclusions: Psychosocial stress may promote the occurrence of MCS, but there was no distinctive psychosocial or behavioral profile of patients related to stress vulnerability. The patients had many physical symptoms and psychiatric comorbidities, but paradoxically, not so many psychological symptoms were complained of.

References :

- 1) Tsujiuchi Y, Kumano H, Yoshiuchi K, Tsujiuchi T, Nakao M, Kuboki T, Okano Y: Psychosomatic investigation on multiple chemical sensitivities. Japanese Journal of Psychosomatic Medicine, 2002;42(3):206-216.[in Japanese]
- 2) Fiedler N, Kipen HM, DeLuca J, Kelly-McNeil K, Natelson B: A controlled comparison of multiple chemical sensitivities and chronic fatigue syndrome. Psychosom Med, 1996; 58(1):38-49

Objectives & Notes

Kenneth Fine, M.D.

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Training:

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Current Faculty Appointments:	Director, Intestinal Health Institute
Medical School/ University Attended	University of Missouri – Kansas City, School of Medicine
Internship:	Baylor University Medical Center, Dallas
Residency:	Baylor University Medical Center, Dallas
Board Certifications:	Internal Medicine, Gastroenterology
Other Information: (including titles of books or articles you have recently written):	Numerous scientific publications on the topics of gluten sensitivity, microscopic colitis, and chronic diarrhea, intestinal physiology.

SPEECH TITLE: “**Treating Systemic Inflammation from an Intestinal Viewpoint**”

At the end of this Presentation, the participant should be able to:

1. Understand the intestine’s role in systemic inflammatory disorders
2. How food antigens and flora are highly immunogenic
3. How to approach an anti-inflammatory diet, intestinal cleansing, and probiotics to minimize immune stimulation from the gut.

The above information was provided by the Speaker.

Objectives & Notes

Sherry Rogers, M.D.

Date of talk: Friday, June 9, 2006, 2:30pm

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Training:

Current Job Description:	Physician/Author
Medical School/ University Attended	S.U.N.Y. Health Sciences Center at Syracuse
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Other Information:	Detoxify or Die, The High Blood Pressure Hoax, No More Heartburn, Pain Free in 6 Weeks, Tired or Toxic, The Cure is in the Kitchen, Wellness Against All Odds, Monthly referenced newsletter Total Wellness and much more.

SPEECH TITLE: “Detoxification for Reversing End-Stage Chronic Diseases, Part II”

At the end of this Presentation, the participant should be able to:

1. Learn that phthalates poison PPAR and peroxisomes, which direct all major fundamental metabolic processes leading to chronic diseases, including arteriosclerosis, chronic inflammation, metabolic syndrome, cancer and more.
2. Phthalate toxicity can be indirectly suspected through numerous metabolic clues, like low DHA/EPA ratio, elevated VLCFA like behenic, lignoceric and hexacosanoic acids, rbc zinc deficiency, elevated lipid peroxides and gene damage like 8-OhdG.
3. Appreciate that using molecular biochemistry assays of human body chemistry followed by detoxification has allowed healing of diseases that are still taught to need chronic medications.

Disclosures:

Author of over a dozen highly referenced books and monthly newsletter on healing of chronic “incurable” diseases, and has received small honorarium from Carlson Laboratories and MetaMetrix Laboratories in the last year for non-CME presentations

The above information was provided by the Speaker.

Detoxification for reversing end-stage diseases, Part II

Sherry A. Rogers, MD, ABFP, ABEM, FACAAI, FACN

Abstract:

The diagnosis of phthalate toxicity begins with identifying the specific tell-tale long chain fatty acid elevation (like behenic, lignoceric, hexacosanoic), imbalance in DHA/EPA ratios, and intracellular zinc deficiency, and abnormal organic acid indicators of damaged fatty acid metabolism (like suberate, adipate and ehtylmalonate) for starters. Because catalase is made in peroxisomes and essential for hydrogen peroxide neutralization, elevated lipid peroxides and 8-OhdG gene damage can also be an indirect indicator of phthalate overload, and are reversible. Then depuration begins after nutrient repair, boosting glucuronidation, as well as discharging phthalates from membranes while simultaneously lowering the load of competing xenobiotics.

Evidence shows the far-reaching effects of poisoned phthalates. They deplete catalase, tocopherols, carnitine, zinc, and DHA while raising reactive oxygen species and lipid peroxides, which in turn promote metastases and fuel all disease. Proof of success is in reversal of symptoms plus normalization of biochemical parameters such as gene markers 8-OhdG and p53, plus lipid peroxides, fatty acids, hs-CRP, fibrinogen and much more as part of this integrative program.

The focus is on correcting nutrient deficiencies and depurating toxicities, since they are the underlying causes of 95% of all disease and even control gene expression, which is responsible for the remaining 5% of disease. The results include reversed Alzheimer's, cancer (metastatic malignant melanoma), end-stage congestive heart failure, cardioversion-resistant atrial fibrillation, multiple chemical sensitivities, intractable seizures, macular degeneration, and hypertension. In medicine these diagnoses mandate a lifetime sentencing to drugs and are considered irreversible, but are actually curable on this evidence-based program. The label is irrelevant, what matters is identifying the true underlying biochemical defects creating the pathologies and then reversing them.

Conclusion: Drugs merely poison malfunctioning pathways, temporarily reducing symptoms but leading to new ones as drugs further deplete detoxification nutrients and foster ignoring repair of causative defects. The beauty of a program to identify biochemical defects is that this corrects the underlying biochemical abnormalities (sometimes dating back to prenatally as recent government studies show) and unloads xenobiotics sufficiently to allow activation of natural healing mechanisms. This program is significantly less expensive, less toxic, and heals versus suppressing symptoms with lifelong expensive drugs, enabling chronic diseases to virtually disappear. It is done at home with non-prescription items under physician guidance, which is the purpose of this presentation. We have the tools for a new generation of physicians trained in not merely "managing" disease, as practice guidelines instruct, but actually curing diseases.

References:

Alonso-Magdalena P, et al, The estrogenic effect of bisphenol A disrupts pancreatic B-cell function in vivo and induces insulin resistance, *Environ Health Perspect* 114:106-12, 2006

Rogers SA, *The High Blood Pressure Hoax*, 2005 (for over 700 more references), prestigepublishing.com

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Objectives & Notes

Mohamed B. Abou-Donia, Ph.D.

Date of talk: Friday, June 9, 2006, 3:45pm

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Board Certifications:	American Board of Toxicology (ABT), and Academy of Toxicological Sciences (ATS)
Other Information: (including titles of books or articles you have recently written):	Book Editor, <i>Neurotoxicology</i> , CRC; Publications: more than 300

SPEECH TITLE: “**Involvement of Tri-ortho-cresyl Phosphate in Aircrew Symptoms**”

At the end of this Presentation, the participant should be able to:

1. Incidents of smoke in the cabin of aircrafts involve tri-cresyl phosphate isomers
2. Although Tri-cresyl phosphates have low acute neurotoxicity, some of them cause organophosphate-induced delayed neurotoxicity (OPIDN)
3. The results of the current study show that these chemicals also cause organophosphate-induced chronic neurotoxicity (OPICN) that me explain the aircrew symptoms

The above information was provided by the Speaker.

Involvement of Tri-*ortho*-cresyl Phosphate in Aircrew Symptoms

Mohamed B. Abou-Donia¹, Larry B. Goldstein², and Ali A. Abdel-Rahman¹

¹Department of Pharmacology and Cancer Biology, ²Department of Medicine (Neurology), Duke University Medical Center, Durham, North Carolina

This study was designed to investigate neurological deficits, related to neuronal cell death, in rats following dermal exposure to tri-cresyl phosphate (TCP) and its constituents, tri-*ortho*-cresyl phosphate (TOCP), tri-*meta*-cresyl phosphate (TMCP), and tri-*para*-cresyl phosphate (TPCP). TCP which contains these three cresyl phosphate isomers is being used as an additive despite acute and chronic neurotoxicity associated with it. Because jet engine oils contain up to 3 percent TCPs as anti-wear agent, exposure to these agents cannot be ruled out. Although the cholinergic neurotoxicity of TCP isomers is low, TOCP is capable of causing organophosphate-induced delayed neurotoxicity (OPIDN). In recent incidents of smoke in the cabins of aircrafts, air crew members and passengers were exposed to tri-cresyl phosphate (TCP) present in contaminated air bleed resulting from leakage of jet engine lubricating oils and hydraulic fluids. As early as 1977, a report documented the incapacitation of an aircraft navigator during flight. Recently, the Association of the Flight Attendants reported 750 incidents that involved 900 flight attendants since 1998. Indeed, a number of air crew members including, pilots and flight attendants have consistently complained of neurological illnesses, such as headache, dizziness, cognitive dysfunction, difficulty concentrating, tremors and generalized weakness; typical symptoms of organophosphate-induced chronic neurotoxicity. Because disturbance of the mental and neuromuscular function of aircraft flight personnel during flight is of great concern, further investigation into the potential neurological deficits from exposure of synthetic oil fumes that are generated during some flights is urgently warranted. Male Sprague-Dawley rats (250-300 g) were randomly divided into control and treated groups (n = 10, each). A daily dose of 2.5 mg/kg of test compounds was applied on the skin of a pre-clipped area on the back of the neck for 30 days. Measures of general toxicity such as feeding behavior, clinical conditions and body weight were recorded. Twenty four hours after the last treatment, the rats were evaluated for behavioral, biochemical and histopathological assessment. The results show that TOCP, TMCP and TPCP caused sensorimotor deficits and cholinesterase inhibition, accompanied by brain neuronal cell death. These results that are consistent with organophosphate-induced chronic neurotoxicity (OPICN), explain the aircrew symptoms associated with smoke in cabins of aircrafts.

24th ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT Schedule

Saturday, June 10, 2006

8:00 **ANNOUNCEMENTS/MODERATOR:** Kaye H. Kilburn, M.D.

8:05 Hiroaki Kumano, M.D., Ph.D., "Symptom Profile of MCS in Actual Life"

8:25 Q & A

8:35 Joaquim Fernandez Sola, M.D., "Multiple Chemical Sensitivity Induced After Organophosphate Exposure"

8:55 Q& A

9:05 Kalpana Patel, M.D., "Autism – ADHD - Heavy Metal Toxicity and Environmental Sensitivity"

9:25 Q & A

9:35 Mohamed B. Abou-Donia, Ph.D., "Gene Expression of the Rat Brain Following acute Sarin Exposure"

9:55 Q & A

10:05 BREAK WITH EXHIBITORS

10:50 William J. Rea, M.D., "Treatment of Chronic Hypersensitivity and Disease: The Hormetic Effects"

11:10 Q & A

11:20 Klaus-Dietrich Runow, M.D., "Detoxification of Heavy Metal Intoxication in Children"

11:40 Q & A

12:00 BUFFET LUNCH WITH THE EXHIBITORS

1:30 **MODERATOR:** Jean Monro, M.D.

1:30 Theodore R. Simon, M.D., "Brain Imaging in 2006"

1:50 Q & A

2:00 Garth Nicolson, Ph.D., "Lipid Replacement and Antioxidant Nutritional Therapy for Restoring Mitochondrial Function and Reducing Fatigue in Chronic Fatigue Syndrome and other Fatiguing Illnesses"

2:20 Q & A

2:30 Bertie Griffiths, Ph.D., "Flow Cytometric Evaluation of Phagocytic Activity of Granulocytes in Whole Blood Against Different Microorganisms in Environmentally Ill Patients"

2:50 Q & A

3:00 BREAK WITH EXHIBITORS

3:45 Panel Discussion/Case Studies: Ron Overberg, Ph.D., "A Case of Mistaken Food Sensitivity"

5:00 AJOURN

SATURDAY, JUNE 10, 2006

ABSTRACTS

AND

HANDOUTS

Objectives & Notes

Hiroaki Kumano, M.D., Ph.D.

Date of talk: Saturday, June 10, 2006, 8:05am

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Other Information: (including titles of books or articles you have recently written):	Symptom profile of multiple chemical sensitivity in actual life. Psychosom Med 67:113-124, 2005; Use of QEESI questionnaire for a screening study in Japan. Toxicol Ind Health 21:113-124, 2005; Application of Quick Environment Exposure Sensitivity Inventory (QEESI) for Japanese population: study of reliability and validity of the questionnaire. Toxicol Ind Health 19:41-49, 2003

SPEECH TITLE: "Symptom Profile of MCS in Actual Life"

At the end of this Presentation, the participant should be able to:

1. Know how to measure the levels of chemical exposure by the wearable device of gas sampling.
2. Know how to measure MCS symptoms with the Ecological Momentary Assessment.
3. Know whether the definition of MCS can be confirmed in actual life.

The above information was provided by the Speaker.

The symptom profile of Multiple Chemical Sensitivity in Actual Life ¹⁾

Hiroaki Kumano

Department of Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo

Goals and objective: This study was conducted to confirm the definition of multiple chemical sensitivity (MCS) in actual life: that multiple symptoms are provoked in multiple organs by exposure to, and ameliorated by avoidance of, multiple chemicals at low levels. We used the Ecological Momentary Assessment ²⁾ to monitor everyday symptoms and the Active Sampling (AS) and Passive Sampling (PS) methods to measure environmental chemical exposure. Our hypotheses of this study were the following: 1) Possible causative chemicals are detected in all but few patients; 2) There are significant differences in not only physical symptoms, but also psychologic symptoms between when MCS patients have episodic symptoms and when they do not. 3) There are no significant differences in any items of physical or psychologic symptoms between patients with no episodic symptoms and control subjects.

Methods: Eighteen patients with MCS, diagnosed according to the 1999 consensus criteria, and 12 healthy controls participated in this study. Fourteen patients and 12 controls underwent 1-week measurement of physical and psychologic symptoms and of the levels of exposure to various chemicals. Each participant was requested to wear the watch-type electronic diary (ED) during the 1-week study period. MCS patients were instructed to respond to the ED once in the morning and once in the afternoon prompted by beeps at random intervals (random prompts) and to answer the same ED questions when they experienced hypersensitivity symptoms that were sufficiently annoying to record (patient-initiated symptom prompts). Controls were instructed to answer the ED four to five times per day in response to beeps at random intervals (random prompts). MCS patients were requested to carry an air sampling pump with two cartridges for carbonyl compounds and VOCs, respectively, for the AS method and two passive samplers for carbonyl compounds and VOCs, respectively, without an air sampling pump for the PS method for 1 week. Controls were asked to carry only two passive samplers for the PS method for 1 week because none had as strong episodic symptoms as were diagnosed with MCS. Linear mixed models were used to test the hypotheses regarding the symptom profile of MCS patients.

Results: Some causative chemicals were detected in 11 of 14 MCS patients. Two other patients did not report any hypersensitivity episodes, whereas passive sampling showed far less exposure to chemicals than in control subjects. Another subject reported episodic symptoms but was excluded from the following analyses because no possible chemical was detected. Eleven of the 17 physical symptoms and all four mood subscales examined were significantly aggravated in the interview based on “patient-initiated symptom prompts.” On the other hand, there were no differences in physical symptoms or mood subscales between MCS patients and control subjects in the interview based on “random prompts.”

Conclusions: MCS patients do not have either somatic or psychologic symptoms under chemical-free conditions, and symptoms may be provoked only when exposed to chemicals.

References:

1) Saito M, Kumano H, Yoshiuchi K, Kokubo N, Ohashi K, Yamamoto Y, Shinohara N, Yanagisawa Y, Sakabe K, Miyata M, Ishikawa S, Kuboki T: Symptom profile of multiple chemical sensitivity in actual life. *Psychosom Med* 2005;67:318-325.

2) Stone AA, Shiffman S. Ecological momentary assessment (EMA) in a behavioral medicine *Ann Behav Med* 1994;16:199–202.

Objectives & Notes

Joaquim Fernandez Sola, M.D.

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SPEECH TITLE: “Multiple Chemical Sensitivity Induced After Organophosphate Exposition”

At the end of this Presentation, the participant should be able to:

1. Know diagnostic criteria of MULTIPLE CHEMICAL SENSITIVITY
2. Consider pathogenic mechanisms leading to MULTIPLE CHEMICAL SENSITIVITY
3. Know common circumstances of organophosphate exposure to avoid development of MULTIPLE CHEMICAL SENSITIVITY

The above information was provided by the Speaker.

Joaquim Fernandez-Solà M.D. Ph.D. Santiago Nogué Xarau*

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AIM: This presentation considers the existence and characteristics of Multiple Chemical Sensitivity (MCS) induced after toxic exposure to insecticide organophosphate agents. I will review the previous medical literature and explain our experience in this field at the Hospital Clinic in Barcelona-Spain.

BACKGROUND: MCS is a pathophysiologically undefined hypersensitivity to all sorts of chemicals at such low concentrations that healthy persons show no discernable reaction. MCS describes a breakdown in prior natural or innate tolerance, like a diabetic's loss of tolerance for sugar. Although its existence has been questioned, the great number of subjects involved with similar repetitive clinical characteristics does not allow to be considered this disorder as an invention. It is more frequent in the last 40 years with the development of petrochemical products nearby. There is no generally accepted case definition for MCS and the majority of instruments available for screening are brief and approach only one or two dimensions of the problem.

DEFINITION: This entity was first defined by Cullen et al (*Occup Med* 1987;2: 655-661) and completed by Eisenberg et al in their report to Congress (Science 1998). Finally a consensus criteria on MULTIPLE CHEMICAL SENSITIVITY was established (*Arch Environ Health* 1999; 54: 147-149) with definition criteria including

1) Acquired chronic condition, 2) With symptoms that recur reproducibly, 3) In response to low levels of exposure, 4) To multiple unrelated chemicals, 5) Which improve or resolve when incitants are removed and 6) Indicating that symptoms occur in multiple organ systems.

EPIDEMIOLOGY and CLINICAL FEATURES: Up to 15-36 % USA population is especially sensitive to certain chemicals, and around 5% fulfill MCS criteria. Women are more sensitive than men to MCS. The most frequent symptoms include: fatigue, neurocognitive disorders, cephalalgia, instability, insomnia, cardiovascular: arrhythmia, hyper or hypotension, Raynaud, respiratory: asthma, reactive airway dysfunction, digestive: colic abdominal pain, dyspepsia, G-E reflux, abdominal distension, ORL: odor sensitivity, odinophagya, Skin: eczema, pruritus, musculo-skeletal: muscle pain and cramps, arthralgia, and diverse gynecologic and urinary symptoms. In spite of the lack of physical illness and absence of pathology, patients often experience extreme disability, because their symptoms are triggered by common environmental exposures

What Sensitivity is NOT: It is not a classical delayed type of hypersensitivity, Allergy, Acute Intoxication, Irritability or anxiety, Personality, Mood, Somatization or Simulation disorder.

BASIS OF MCS: Numerous theories have been suggested to explain the condition that encompasses immunotoxic, behavioral, psychiatric, iatrogenic and sociologic mechanisms. MCS supposes a loss of tolerance to toxic chemical agents with predisposition towards increased sensitivity in certain persons similar to that proposed by the enzyme polymorphism model of toxicology. There is a failure in the classical toxicological dose-effect relation. The definition of MCS is based on clinical criteria, without no useful laboratory or diagnostic imaging marker. Psychological and psychosomatic hypotheses have been proposed, with 2% of the cases presenting previous mood disorders and 37% longstanding mood disorders after MCS.

TOXIC EFFECTS OF ORGANOPHOSPHATES: include 3 main aspects: 1) An ACUTE: CHOLINERGIC Syndrome with Muscarinic (bradycardia, myosis, sialorrhoea, vomiting, diarrhea, sweating) or Nicotinic (muscle cramps and weakness, fasciculations) symptoms. 2) A SUBACUTE MUSCULAR Syndrome, and 3) CHRONIC POLYNEUROPATHY or –NEUROCOGNITIVE SYNDROME – OPICN-(organophosphorous-induced chronic neurotoxicity) syndromes.

PRODUCTS AND EXPOSITION: MCS of toxic origin involves different products, with Pesticides (Organophosphates, Pyrethrin), Solvents (Phenol, Xylol), Chlorinate compounds, Glutaraldehyde, Hydrocarbons, Fuel or Combustion products or combination exposures being the most frequent. MCS may start after single high-level toxic exposure or chronic low-dose exposure. In 1991, Ashford & Millar established a MCS Classification in four

types: 1) Isolated cases with heterogeneous exposure usually at low-chemical level at home or the workplace; 2) Occupants of sick buildings, usually women in the workplace; 3) Industrial workers usually men with acute high-dose toxic exposure, and 4) Contaminated air and water in the community. Use of environmental pesticides or contaminants. In previous literature, isolated cases of MCS have been reported following agricultural, industrial and accidental exposure to toxic products.

PROSPECTIVE STUDY: (Fernandez-Solà J et al. *Chronic Fatigue Syndrome and Multiple Chemical Hypersensitivity after insecticide exposition Medicina Clinica (Bar) 2005;124:451-453*).

During a period of 4 years (2000-2003) we attended in the Toxicology Unit of the Hospital Clinic (Barcelona, Spain). Thirty nine in 273 patients (14%) had been exposed to pesticides. In these patients we performed clinical and toxicological anamnesis considering 1) Acute mucosa irritative syndrome, 2) Muscarinic / Nicotinic manifestations, 3) Neurocognitive impairment 4) Gastrointestinal manifestations. 5) Muscle pain / Fibromyalgia, 6) Physical or Mental Fatigue, and 6) Signs of sensitivity disorder. MCS was defined as a Consensus Document. We specifically made an assessment of the type of plaguicide they had been exposed, to the circumstances of exposure (in the workplace, accidental, and whether exposure was a single event or repetitive, the method of plaguicide use (contact, environmental dispersion or fumigation) and the period between fumigation and patient's contact. Finally, blood cholinesterase activity analysis was determined.

In all patients with sensitivity we applied the QEESI Questionnaire. (Miller C et al. *Toxicol Ind Health 1999; 15:370-385*), which evaluates 5 different scales: 1) Intolerance to inhalatory chemical products (score 0-100, with the threshold being < 20); 2) Intolerance to non-inhalatory chemical products (score 0-100, threshold < 12); 3) Severity of symptoms (0-100, threshold < 20); 4) Masked or non-apparent chemical products (0-100), and 5) They were also given an impact sensitivity scale on daily activity (0-10). The global score may range from 0 – 420 points. In addition, Quality of life was evaluated with the use of SF-36, Daily Functional Impairment 0with (H.A.Q), and the presence of comorbidity was evaluated.

RESULTS: Of the 39 subjects exposed, nine cases (26%) fulfilled MCS criteria. All were women with a mean age of 43 ± 7 years. The type of Plaguicide was organophosphate (chlorpyrifos and dychlorvos) in 5 cases, organophosphate and pyrethroids in 3 cases and pyrethrums in one case. In 6 cases (66%) there was an epidemic outbreak with a mean of 4.2 cases. The period between fumigation and exposure was of less than 24 h in 4 cases (44%). The origin was at the workplace in 8 cases (hotels, administrative offices, health centers and shopping centers) and accidental in one case (manufacturing pesticides).

All nine patients with MCS presented acute mucosa irritative syndrome, chronic fatigue syndrome (6 to a moderate degree), and also neurocognitive syndrome. Fibromyalgia was present in 6 (67%), and gastrointestinal manifestations in 2 patients (22%). None presented manifestations of acute organophosphate intoxication, and cholinesterase levels were also normal in all of them. Most of these patients presented comorbidities such as sicca syndrome, asthma, irritable bowel syndrome and dysthymia.

At 2.5 years of follow-up, one third of these patients had permanent working disability, that increased to two-thirds at five-years follow-up. Only one patient 11% presented a self-limited process (< 1 year).

After this study, we recruited 51 patients with MCS, the majority of whom are women (mean age 45 ± 5 years), with clear previous toxic exposure in 95%. Comorbidity is frequent, and half of them have developed a persistent disabling disease.

PATHOGENESIS: In MCS, the main Predisposing factors are: 1) Cholinergic hypersensitivity, 2) Paroxonase (PON) or Cholinesterase (ChE) gene polymorphism, and 3) Differences in permeability of the brain barrier. Triggering factors induce an acute increase in cholinergic activity, with dysregulation mechanisms, with MCS being clinically apparent. Perpetuating factors maintain disease activity over time.

Common pathogenic pathways between OP exposure and development of MCS imply the development of cholinergic hypersensitivity, observed in clinical and experimental studies (Oberstreet and Djuric. *Ann NY Acad Sci 2001; 933; 92-102*). Behavioral characteristics such as abnormal sleep, activity, and appetite may suggest subclinical MCS. Peripheral tissues such as airway and intestinal smooth muscle appear to be more sensitive. Some authors have suggested a synthesis of biologic and neuropsychologic mechanisms.

Disrupted mechanisms in MCS include: 1) Inhibition of vascular cholinesterase with secondary increased cholinergic activity, 2) Disturbed GABA_A receptor activity (*Corrigan, Med Hypoth 1994; 43: 195*), 3). Increased neuronal oxidative stress, 4) Elevated Nitric Oxide, 5) Disturbed NMDA glutamate receptors, and 6) Increase neuronal Calcium-load leading to apoptosis. (*Abou-Donia MD. OP induced chronic neurotoxicity. Arch Environ Health 2003;58: 484*):

The main pathogenic mechanism explaining the development of MCS is **Toxic-induced loss of tolerance (TILT)**. This implies 1) The loss of tolerance in susceptible people after exposure to several toxics, 2) The subsequent development of symptoms at a very low level exposure to the original chemical or other non related chemicals isolated or combined. Other phenomenon which may also develop in this process are 1) ABDICTION, a biphasic evolution of symptoms (stimulation and deprivation-abstinence); 2) MASKING: where some responses to chemicals are not evident (Addiction, Apposition and Habituation phases); 3) KINDLING where low dose repetitive exposure causes increased excitability in neurons located at limbic NCS area. Stimulus is reinforced with unpleasant threshold for multiple types of sensory stimuli; 4) SPREADING, with propagation once started with similar response to low dose of other agents structurally unrelated.

CONCLUSIONS: 1) Pesticide exposure (Organophosphates, Pyrethrums) may trigger Multiple Chemical Sensitivity in one fourth of exposed subjects. 2) Exposure is usually low-dose repetitive, but also acute high-dose exposure may trigger MCS. 3) In most cases MCS is persistent and disabling and co-morbidities including the Chronic Fatigue Syndrome, Fibromyalgia, Sicca syndrome are frequent. 4) Correct Prevention measures may avoid the development of most cases. 5) However, fulfillment of fumigation safety rules is necessary to avoid increasing MCS of toxic origin, and chemical threshold limit values should be revised.

Objectives & Notes

Kalpana Patel, M.D.

Date of talk: Saturday, June 10, 2006, 9:05am

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SPEECH TITLE: “Autism – ADHD - Heavy Metal Toxicity and Environmental Sensitivity”

At the end of this Presentation, the participant should be able to:

1. Role of heavy metal toxicity in Autism and Hyperactivity
2. Synergistic role of lead toxicity with mercury toxicity in Autistic children.

The above information was provided by the Speaker.

Objectives & Notes

Mohamed B. Abou-Donia, Ph.D.

Date of talk: Saturday, June 10, 2006, 9:35am

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Medical School/ University Attended	University of California, Berkeley
Board Certifications:	American Board of Toxicology (ABT), and Academy of Toxicological Sciences (ATS)
Other Information: (including titles of books or articles you have recently written):	Book Editor, <i>Neurotoxicology</i> , CRC; Publications: more than 300

SPEECH TITLE: “Gene Expression of the Rat Brain following acute Sarin Exposure”

At the end of this Presentation, the participant should be able to:

1. Sarin causes three neurotoxic actions: 1) acute, 2) OPIDN and 3) OPICN.
2. Sarin causes alterations in brain gene expression that persists for long time.
3. Sarin-induced gene expression explains the known neurotoxic actions caused by sarin exposure.

The above information was provided by the Speaker.

Gene Expression of the Rat Brain following acute Sarin Exposure

Mohamed B. Abou-Donia¹, Tirupapuliyyur V. Damodaran¹, Anand G. Patel¹, Stephen T. Greenfield¹, Holly K. Dressman², Simon M. Lin³, and Department of Pharmacology and Cancer Biology¹, Department of Molecular Genetics and Microbiology², Department Biostatistics and Bioinformatics³, Duke University Medical Center, Durham, North Carolina

Sarin was developed during World War II as a nerve agent (nerve agent GB). Sarin has three neurotoxic actions. First, the irreversible inhibition of acetylcholinesterase (AChE) leading to the accumulation of acetylcholine (ACh) and subsequent over-stimulation of the nicotinic and muscarinic receptors resulting in cholinergic effects and death at high exposure. Second, a delayed onset of ataxia accompanied by Wallerian-type degeneration of the axon and myelin of the central and peripheral nervous systems, known as organophosphorus ester-induced delayed neurotoxicity (OPIDN). Third, a long-term neurological deficits accompanied by brain neuronal cell death that has been characterized recently as organophosphorus ester-induced chronic neurotoxicity (OPICN). We have studied sarin-induced global gene expression patterns for an early time point (15 minutes: 0.5 X LD₅₀) and late time point (3-months: 1 X LD₅₀) using affymetrix: Rat Neurobiology U34 chips in male, Sprague- Dawley rats. We have identified a total number of 65 and 38 genes showing statistically significant alterations from control levels at 15 minutes and 3-months, respectively. At the early time point, the genes that were more in number than any other group are classified as ion channels, cytoskeletal and cell adhesion molecules, as well as neuropeptides and their receptors. Other groups included cholinergic signaling, calcium channels and binding proteins, transporters, chemokines, GABAnergic, glutamatergic, aspartate, catecholaminergic, nitric oxide, purinergic, and serotonergic signaling molecules. At the late time point, the genes that were more in number than any other group are classified as calcium channels and binding proteins, cytoskeletal and cell adhesion molecules, and GABAnergic signaling molecules. Seven molecules (Ania-9, Arrb1, CX-3C, Gabab-1d, Nos2a, Nrnx 1b, Pde2) showed persistent altered expression in both time points. Selected genes from each of these time points were further validated using semi quantitative RT-PCR approaches. Some of the genes that were identified in the present study have been shown to be involved in organophosphate-induced neurotoxicities by other groups as well as ours. Principal component analysis of the expression data from both time points was used for comparative analysis of the gene expression, which indicated that the changes in gene expression were function of dose and time of euthanasia after the treatment. Our model also predicts that besides dose and duration of post-treatment period, age and possibly other factors may be playing important roles in the regulation of pathways, leading to development of neurotoxicity. (Supported, in part, by the U.S. Army Medical and Materiel Command Center under contract project order DAMD 17-99-1-9020).

Objectives & Notes

William J. Rea, M.D.

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Board Certifications:	American Board of Surgery, American Board of Thoracic Surgery, American Board of Environmental Medicine
Other Information: (including titles of books or articles you have recently written):	Optimum Environments for Optimum Health and Creativity (book); Averse Health Effects of Indoor Molds.

SPEECH TITLE: “**Treatment of Chronic Hypersensitivity and Disease: The Hormetic Effects**”

At the end of this Presentation, the participant should be able to:

1. To understand the hormesis principle in treatment modalities.
2. To understand and apply the hormetic effect to treatment.
3. To understand how hormesis applies to provocation neutralization.

The above information was provided by the Speaker.

TREATMENT OF CHRONIC HYPERSENSITIVITY AND DISEASE: THE HORMETIC EFFECTS

William J. Rea, M.D., F.A.S.C., FA.A.E.M.

The treatment of chemical sensitivity and chronic degenerative disease is enhanced by the knowledge of the hormetic effect. An example of how change in dose can change treatment is that one level may result in a stimulating effect while another may result in inhibitory phenomena. The technique of intradermal provocative neutralization easily demonstrates the hormetic principle and how it benefits treatment.

Conclusion: Hormetic principle should be included when formulating treatment protocols.

References:

Calabrese, E.J. and Baldwin, L.A., Special Issue on hormesis, *Hum. Exp. Toxicol.*, 19(1), 2-97, 2000.

Calabrese, E.J. and Baldwin, L.A., A quantitatively based methodology for the evaluation of chemical hormesis, *Hum. Ecol. Risk Assess.*, 3(4), 545-554, 1997.

Objectives & Notes

Klaus-Dietrich Runow, M.D.

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Other Information:	Books: Klinische Oekologie (Clinical Ecology) and Nervenschutz durch Entgiftung (Nerve Protection by Detoxification). First honorary member of the German Society for Environmental and Human Toxicology; The society for Threatened Peoples of Goettingen, Germany, brought me to Kosovo to test for toxic heavy metals in refugee camps near Mitrovica

SPEECH TITLE: “**Detoxification of Heavy Metal Intoxication in Children**”

At the end of this Presentation, the participant should be able to:

1. Estimate the first results and effectiveness of the detoxification treatment regarding the first group of highly lead contaminated children out of the refugee camp in Mitrovica/Kosovo. The therapy started in April 2006.

The above information was provided by the Speaker.

Detoxification of Heavy Metal Intoxication in Children

Klaus-Dietrich Runow

Speech #2 Saturday, June 10, 11.20 am

Since they were burned out of their homes during the Kosovo war in 1999 a group of more than 500 Roma people living at the edge of the derelict Treпча mines near Mitrovica / North Kosovo. The toxic load comes from the slag heaps that waft clouds of heavy metal containing dust into the air, water and soil. Over 30 people died during the last six years. Medical investigations showed extreme high levels of heavy metals. In 2004 WHO conducted blood tests on several children in the camps after a four-year-old Romani girl died of lead poisoning. All children tested had dangerously high lead levels. Although the WHO recommended immediate evacuation the people still live in the polluted area.

Medical treatment for the first highly contaminated patients out of the refugee camp started in April 2006 in our clinic in Bad Emstal/Germany. The lead level in the hair of the 7-year-old boy Denis was 1.200 times over the reference range. He will be treated together with his father and sisters. First observations and results regarding the effectiveness of the detoxification therapy will be discussed.

Klaus Dietrich-Runow, Institute for Functional and Environmental Medicine (IFU)

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Special thanks to Society for Threatened People (GfbV), Goettingen, Germany, www.gfbv.de : Tilman Zülch, Jasna Causevic, Frank Witte and Paul Polansky who is living in Kosovo and has been struggling since over 6 years for the evacuation of the polluted camps.

The Detoxification therapy will be supported through the program “A heart for children” from BILD hilft e.V. (a non-profit organization connected to the German newspaper BILD). Special thanks: Frau Martina Krueger und Frau Kottusch.

Objectives & Notes

Theodore R. Simon, M.D.

Date of talk: Saturday, June 10, 2006, 1:30pm

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Training:

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Medical School/ University Attended	Yale
Internship:	University of Rochester
Residency:	Yale and University California at San Francisco
Board Certifications:	ABNM
Other Information: (including titles of books or articles you have recently written):	See CV at www.theodorersimon.com

SPEECH TITLE: **“Brain Imaging in 2006”**

At the end of this Presentation, the participant should be able to:

1. Understand the role of brain scintigraphy in neurotoxic patients.
2. Understand the effects of time on brain function.

The above information was provided by the Speaker.

Brain Imaging in 2006

by Theodore R. Simon, M.D.

Goals and Objectives

This presentation uses a recent pilot study to discuss the role of triple SPECT brain scintigraphy in assessing and following patients with neurotoxic exposure. The presentation seeks to provide a framework for developing rational and efficient algorithms for managing these patients.

This pilot study tested the hypothesis:

*Patients who clinically improved after therapy for neurotoxicity
will have a reversal in abnormalities seen by brain scintigraphy.*

Outline

Various qualitative and quantitative strategies were used to assess tracer distributions within the brain. These were then tested against both qualitative and quantitative clinical parameters to determine the relationship between clinical and scintigraphic measures of changes in the neurotoxic patient during the interval between scintigraphic examinations. Ten patients were included in this pilot study. The six women and four men had two SPECT brain examinations separated by 5 to 62 months (average \pm SEM=1.93 \pm 0.3 years). The appearance of SPECT brain examinations performed failed to improve longitudinally, despite documented clinical success.

Conclusions

The results showed that glutathione-mediated functional impairment continues--and generally progressively worsens--despite clinical success in treating the patient. Patients who were substantially restored to productive neurological function remained impaired in their ability to detoxify their brains.

Objectives & Notes

Garth Nicolson, Ph.D.

Date of talk: Saturday, June 10, 2006, 2:00pm

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Training:

Current Job Description:	President & Chief Scientific Officer, Research Professor, The Institute for Molecular Medicine, Huntington Beach, California
Current Faculty Appointments:	President & Chief Scientific Officer, Research Professor, The Institute for Molecular Medicine, Huntington Beach, California, Cojoint Professor, Faculty of Science and Technology, University of Newcastle (Australia), Professor of Integrative Medicine, Capital University of Integrative Medicine, Washington, DC.
Medical School/ University Attended	University of California, San Diego, Ph.D. 1970 Biochemistry & Molecular Biology
Board Certifications:	Assoc. Member AAEM, Member AACR, Member and Founder, Metastasis Research Soc.
Other Information: (including titles of books or articles you have recently written):	Published over 575 medical and scientific papers, edited 14 books, served on the Editorial Boards of 20 medical and scientific journals. A Colonel (Honorary) of the U.S. Army Special Forces and a U.S. Navy SEAL (Honorary) for his work on Armed Forces and veterans' illnesses.

SPEECH TITLE: "Lipid Replacement and Antioxidant Nutritional Therapy for Restoring Mitochondrial Function and Reducing Fatigue in Chronic Fatigue Syndrome and other Fatiguing Illnesses"

At the end of this Presentation, the participant should be able to:

1. Understand the relationship between fatigue and mitochondrial function
2. Understand how mitochondrial membrane damage can be repaired by lipid replacement and antioxidant therapy
3. Use lipid replacement/antioxidant therapy in their clinical practice

The above information was provided by the Speaker.

Lipid Replacement and Antioxidant Therapy for Restoring Mitochondrial Function in Fatigue and Fatiguing Illnesses

Garth L. Nicolson, Rita Ellithorpe and Robert Settineri
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Decreased mitochondrial function and loss in the efficiency of the electron transport chain are related to aging and fatigue [1,2]. Lipid Replacement Therapy (LRT) along with antioxidants can circumvent ROS membrane damage and replace and restore mitochondrial and other cellular membrane functions via delivery of replacement lipids in their unoxidized, undamaged states [1]. Recent clinical trials have shown the benefit of LRT plus antioxidants in restoring mitochondrial electron transport function and reducing fatigue [3,4]. In aging subjects mitochondrial function was restored to levels found in young adults in consort with reductions in fatigue, suggesting the anti-aging and anti-fatigue benefits of LRT plus antioxidants in protecting mitochondrial and other cellular membranes from oxidative and other damage and preventing loss of function. In a clinical study we determined if mitochondrial function is reduced in aging subjects with mild to moderately severe fatigue, and if this can be reversed with a nutritional supplement (NTFactor) that replaces damaged mitochondrial lipids [5]. Participants (n=26) with mild to moderate fatigue (Piper Fatigue Scale, PFS [6]) were admitted when their fatigue could not be explained. Blood leukocytes were analyzed for mitochondrial function using Rhodamine-123. Subjects (mean age 68.9±7) completed 12 weeks on product, and 16 also completed a wash-out study for an additional 12 weeks. There was a time-dependent reduction in overall fatigue in ten moderately fatigued subjects (PFS 5.75±0.62, range 4.09-8.45) while on supplement but not in eleven mildly fatigued subjects (PFS 1.42±0.2, range 1.0-2.55). After four weeks the average score of moderately fatigued subjects was reduced to 4.59 (20.2% reduction, p<0.005), but there was no significant change in mildly fatigued subjects. Further use for a total of eight or 12 weeks decreased the moderately fatigued subjects to 3.80±0.41 (33% reduction, p<0.001) or 3.71±0.48 (35.5% reduction, p<0.001), whereas the mildly fatigued subjects were not significantly different. Analysis of mitochondrial function indicated that four and eight weeks of NT Factor use in moderately fatigued subjects increased function by 15% and 26.8%, respectively, and restored mitochondrial function to levels similar to those found in young adults. Further use for a total of 12 weeks did not cause a further increase in function. Some subjects were monitored 12 weeks after discontinuing the supplement. Their fatigue and mitochondrial function were found to be intermediate between the initial results and at eight weeks indicating that continued use of NTFactor is necessary to maintain lower fatigue scores and mitochondrial function. In another study we examined Chronic Fatigue Syndrome and Fibromyalgia Syndrome patients and also found that NTFactor reduced fatigue and increased mitochondrial function [7].

References

2. Nicolson GL. Lipid replacement as an adjunct therapy in chronic fatigue, anti-aging and restoration of mitochondrial function. *JANA* 2003; 6(3):22-28.
3. Paradies G, Petrosillo G, Pistolese M, Ruggiero F. Reactive oxygen species affect mitochondrial electron transport complex I activity through oxidative cardiolipin damage. *Gene* 2002; 286:135-141.
4. Ellithorpe RR, Settineri R, Nicolson GL. Reduction of fatigue by use of a dietary supplement containing glycopospholipids. *JANA* 2003; 6(1):23-28.
5. Agadjanyan M, Vasilevko V, Ghochikyan A, Berns P, Kesslak P, Settineri RA, Nicolson GL. Nutritional supplement (NTFactor) restores mitochondrial function and reduces moderately severe fatigue in aged subjects. *J Chronic Fatigue Syndr* 2003; 11(3): 23-36.
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8. Nicolson GL, Ellithorpe E. Lipid replacement and antioxidant nutritional therapy for restoring mitochondrial function and reducing fatigue in Chronic Fatigue Syndrome and other fatiguing illnesses. *J Chronic Fatigue Syndr* 2006; 13(1) in press.

Objectives & Notes

Bertie Griffiths, Ph.D.

Date of talk: Saturday, June 10, 2006, 2:30pm

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Training:

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Medical School/ University Attended	M.S., B.S., University of Wisconsin, Bacteriology; Ph.D. University of West Indies, Virology
Post Doctoral:	University of West Indies

SPEECH TITLE: "Flow Cytometric Evaluation of Phagocytic activity of Granulocytes in Whole Blood, Against Different Microorganisms in Environmentally Ill Patients"

At the end of this Presentation, the participant should be able to:

1. Be Conversant with the flow cytometer as offering a quantitative phagocytic profile.
2. Associate a variety of illnesses with a defective phagocytic cascade.
3. Appreciate that clinical diagnosis and treatment can be generated from a phagocytic profile.

The above information was provided by the Speaker.

Flow Cytometric Evaluation of Phagocytic Activity of Granulocytes in Whole Blood Against Different Microorganisms in Environmentally Ill Patients

Bertie G. Griffiths, Ph.D.
Environmental Health Center - Dallas, TX

Phagocytosis is an essential protective, universal response of the body against infection. It involves ingestion and killing of invasive microorganisms, and digestion of alien cells and other foreign particles.

The Phagocytic process utilizes two major phagocytes, polymorphonuclear neutrophils (PMN'S), and macrophages. PMN'S are utilized in this investigation.

The Flowcytometric process used in this investigation involve:

1. Labeling with fluorescein isothiocyanate (FITC), the prominent bacteria and fungi isolated from environmentally ill patients, with varied clinical manifestation seen at the Environmental Health Center – Dallas.
2. Phagocytose with neutrophils from patients' peripheral blood.
3. DNA staining with propidium iodide for cytometric discrimination of bacteria and platelet aggregates.
4. Flow cytometric analysis on the Coulter Cytomics FC 500 flow cytometer. By combining fluorescence and side scatter light, phagocytes containing ingested microorganisms are identified.
5. Quantitative phagocytic results were measured fluorometrically, assisted by using the commercial phagotest kit (orpegen pharma).
6. From a profile of neutrophil Phagocytic function, quantitative measurement of cellular response to mitogens or growth factor, pathogenesis and treatment of patients can be generated.

References

Bassoe, C.F. 1984. Flow cytometric studies on phagocyte function in bacterial infections. Acta path. Microbial. Immunol. Scand. Sect. C, 92:167-171.

Robinson, J.P., Carter, W.O. 1993. Flow cytometric analysis of granulocytes. In: Bauer, K.D. et al (eds). Clinical Flow Cytometry, principles and applications. Williams & Wilkins, Baltimore, pp. 405-433.

Objectives & Notes

Ron Overberg, Ph.D., R.D., L.D.

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Board Certifications:	Board Certified Clinical Nutritionist
Other Information: (including titles of books or articles you have recently written):	2005 Revision of the "Rotational Bon Appetit Cookbook;" 2005 "EHC-D Food Rotation and Supplement Guide" Handout

SPEECH TITLE: "A Case of Mistaken Food Sensitivity"

At the end of this Presentation, the participant should be able to:

1. To help patients sort out food reactions
2. To look up the nutritional content of foods

The above information was provided by the Speaker.

24th ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT Schedule

Sunday, June 11, 2006

8:15 **ANNOUNCEMENTS/MODERATOR:** Doug Seba, Ph.D.

8:30 Kaye H. Kilburn, M.D., “Neurobehavioral and Pulmonary Functions in 105 Adults Exposed to Molds and 100 Exposed to Chemicals”

8:50 Q & A

9:00 Kou Sakabe, M.D., “Chemical Sensitivity and Oxidative Stress”

9:20 Q & A

9:30 Jonathan Fox, M.D., “Reversing MCS symptoms with Patient Centered Approach”

9:50 Q & A

10:00 BREAK

10:30 Martha Stark, M.D., “Reversibility of Hypersensitivity and Degenerative Disease: Mechanisms”

10:50 Q & A

11:00 Nancy A. Didriksen, Ph.D., “Neurocognitive Effects of Toxigenic Molds”

11:20 Q & A

11:30 William J. Meggs, M.D., Ph.D., “Toxicity of Chronic Low-Dose Organophosphate Exposures”

11:50 Q & A

12:00 SUMMARY AND CLOSE: Doug Seba, Ph.D.

SUNDAY, JUNE 11, 2006

ABSTRACTS

AND

HANDOUTS

Objectives & Notes

Kaye H. Kilburn, M.D.

Date of talk: Sunday, June 11, 2006, 8:30am

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Training:

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Residency:	Utah, Duke, University of London
Board Certifications:	American Board of Internal Medicine, American Board of Preventive Medicine - Occupational Health
Other Information: (including titles of books or articles you have recently written):	Books: Chemical Brain Injury, Endangered Brains, Molds and Mycotoxins. Archives of Environmental Health: Onboard insecticide use of flight attendants, Affects of chlorine and other chemicals byproducts, including mold and mold products, in brain and lung performance. Archives of Environmental Health

SPEECH TITLE: "Neurotoxicity of Mycotoxins and Other Chemicals"

At the end of this Presentation, the participant should be able to:

1. Recognize mold/mycotoxins as neuro-toxic chemicals
2. Know the comparative impairments of exposures to molds and other chemicals
3. Appreciate why removal from exposure is imperative to save brain function

The above information was provided by the Speaker.

Objectives & Notes

Kou Sakabe, M.D.

Date of talk: Sunday, June 11, 2006, 9:00am

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Training:

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Current Faculty Appointments:	Professor of Environmental Medicine, Kitasato University
Medical School/ University Attended	Tokai University School of Medicine
Internship:	Tokai University Hospital
Residency:	Tokai University Hospital
Board Certifications:	The Japanese Society for Clinical Ecology
Other Information: (including titles of books or articles you have recently written):	Kinases and Phosphatases in Lymphocyte and Neuronal Signaling. Springer-Verlag

SPEECH TITLE: "Chemical Sensitivity and Oxidative Stress"

- 1) Goals and objectives:** It is well known that oxidative metabolites contribute to multi-organ dysfunction and are involved in the pathogenesis and development of environmentally-triggered diseases, such as chemical sensitivity (CS). The aim of this study was to provide evidence for enhanced oxidative stress in CS.
- 2) Outline of talk/abstract:** a) We found significant differences in distribution of genotype frequencies of the GSTP1 gene between patients with chemical sensitivity and healthy controls. b) We found that the PAO levels were significantly decreased in CS patients if compared to healthy donors.
- 3) Conclusion of what is to be learned: References:** a) The GST polymorphism might affect the development of chemical sensitivity or might be linkage disequilibrium by other mutations around this gene that might be association with CS. b) The findings indicate that reduced antioxidant capacity is associated with the extent and severity of CS. c) These observations may assist in providing more information as to how oxidative stress may predispose to CS and suggest attractive therapeutic strategies in the prevention and treatment of CS.
- 4) References:**
 - Y.Matsuzaka et al., Mammalian Genome, 2006, in press
 - S.Hojo et al., Toxicol. Industrial Health., 21:113-124, 2005.
 - E.Straface et al., FEBS Let., 579:2759-2766, 2005.
 - C.Vassalle et al., J. Int. Med., 256:308-315, 2004.

The above information was provided by the Speaker.

Chemical Sensitivity and Oxidative Stress

Kou Sakabe, M.D., Ph.D.

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Part 1 Association of glutathione S-transferase P1 (GSTP1) and not cytochrome P450 1A1 (CYP1A1) polymorphisms with chemical sensitivity in the Japanese population

This study examined the association between the xenobiotics genes *CYP1A1* and *GSTP1* and chemical sensitivity in the Japanese population. Sixty patients with chemical sensitivity and 131 healthy controls were enrolled in this study. We examined the prevalence of three genetic polymorphisms of *CYP1A1* and two of *GSTP1* genes. However, no statistically significant differences in genotype or allele frequency distributions of three genetic polymorphisms of the *CYP1A1* gene were found between patients and controls. Although no significant difference was found in genotype or allele frequency distributions of polymorphism in exon 5 of the *GSTP1* gene, the frequency of T/C genotype in exon 7 of this gene was significantly higher in patient with chemical sensitivity than in normal controls ($P=0.034$). We therefore conclude that heterozygosity for polymorphism of exon 7 in *GSTP1*, but not exon 5 polymorphism in *GSTP1* or three *CYP1A1* polymorphisms, confers susceptibility to chemical sensitivity, though undiscovered causative variants might exist around the site of polymorphism of exon 7 of the *GSTP1* gene.

Part 2 Oxidative imbalance changes as peripheral blood biomarkers of chemical sensitivity

Biomarkers of oxidative stress in peripheral blood from patients with chemical sensitivity were analyzed. Fifty-seven CS patients were recruited. The total antioxidant power (PAO) in peripheral blood was evaluated. We found that the PAO levels were significantly decreased in CS patients if compared to healthy donors (no chemically sensitive group). These results suggest that oxidative imbalance in the peripheral blood of CS patients could major oxidative changes previously described in the central or peripheral nervous system.

Objectives & Notes

Jonathan Fox, M.D.

Date of talk: Sunday, June 11, 2006, 9:30am

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Training:

Current Job Description:	Physician – patient care Nova Scotia Environmental Health Centre
Current Faculty Appointments:	Department of Family Medicine – Dalhousie University
Medical School/ University Attended	Dalhousie
Internship:	University of Ottawa
Residency:	University of Ottawa – Family Medicine
Board Certifications:	Family Medicine

SPEECH TITLE: “Reversing MCS symptoms with Patient Centered Approach”

At the end of this Presentation, the participant should be able to:

1. Recognize most common presenting symptoms in multiple chemical sensitivity (MCS).
2. Understand how symptoms can improve with treatment.
3. Recognize the value of holistic case for the individual with MCS.

The above information was provided by the Speaker.

Reversing MCS Symptoms with Patient Centred Approach

Jonathan Fox¹, Tara Sampalli¹ and Roy Fox¹

¹ Nova Scotia Environmental Health Centre

The Nova Scotia Environmental Health Centre is a medical facility for treating environmentally related illnesses including Multiple Chemical Sensitivities, Chronic Fatigue Syndrome and Fibromyalgia. The care system that has been developed is holistic. The holistic approach is in line with the twelve determinants of health as described by Health Canada. Treatment may include physiotherapy, desensitization therapy, intravenous therapy and counseling. Therapy is also directed towards life style changes through education and group intervention approaches. Mind and body intervention treatment are also offered such as Body Mind Awareness Program, Craniosacral therapy, therapeutic touch and guided imagery. Patients are coached in life style changes, which are aimed at improving overall health, reducing the impact of various symptoms and improving the self-management of the health problems. A study conducted at the Centre that looked at the prevalence of major symptoms in 351 patients at the Centre identified that general symptoms such as difficulty concentrating, fatigue, forgetfulness, and irritability dominated the overall prevalence of symptoms since the start of their illness. Those related to irritation such as headaches, stuff or full sinuses, itchy or burning eyes, and hoarseness or loss of voice were more common after exposure to environmental irritants. A study that was recently completed at the Centre has offered some preliminary evidence that patients at the Centre show improvement in their symptoms and overall health with treatment. An abbreviated version of a symptoms questionnaire, based on the Toronto Health Survey Questionnaire that every patient answers at the time of enrolment was used to measure patient health progress. A total of 182 active patients under the following categories: 6 – 1 yr of treatment at the Centre, 1-2 years of treatment at the Centre, 2+ to a maximum of 5 years of treatment at the Centre and 109 discharged patients participated in the study. Patients showed statistically significant improvement in their overall health under categories such as health since ill 0.05 (6m-1yr), <0.0001 (1-2yr, 2+ years, discharged); too ill to do housework 0.05 (6m-1yr), 0.008 (1-2years), <0.0001 (2+ and discharged); limit contact with people to avoid exposures 0.9 (6m-1yr), 0.09 (1-2yrs), 0.02 (2+ yrs) and <0.0001 (discharged). There were also significant changes in some of the top reported symptoms. Some symptoms showed early improvement such as difficulty concentrating 0.01 (6m-1yr), 0.003 (1-2yrs) and <0.0001 (2+ and discharged); stuffy or full sinuses; headaches 0.9 (6m-1yr), 0.004 (1-2yrs) and <0.0001 (2+ and discharged). Others showed slower and significant changes such as fatigue 0.5 (6m-1yr), 0.3 (1-2yrs), <0.0001 (2+ and discharged); hoarse or loss of voice 0.8 (6m-1yr), 0.5 (1-2yrs), <0.0001 (2+ and discharged). There were other symptoms that did not show significant changes with treatment but were in the top symptoms reported by patients such as trouble seeing at night 0.3 (6m-1yr), 0.2 (1-2yrs), 0.1 (2+ yrs) and 0.9 (discharged); bruise easily 0.6 (6m-1yr), 0.1 (1-2yrs), 0.9 (2+ yrs) and 0.01 (discharged). The results presented are preliminary and would need further inquiry with support from qualitative interviews and a control group to confirm our findings.

REFERENCE

1. Bartha L, Baumzweiger W, Buscher D S et al.1999. Multiple Chemical Sensitivity: A 1999 Consensus. Archives of Environmental Health. **54**: 147-149.
2. Fox R 2002. The Environment and Multiple Chemical Sensitivity. Indoor air 2002, Proceedings
3. Hu H, Stern A, Rotnitzky A, Schlesinger L, Proctor S, Wolf J 1999. Development of a brief questionnaire for screening for multiple chemical sensitivity syndrome. Toxicology Ind. Health. 15(6), 582-588.
4. Joffres M.R., Williams T, Sabo B, Fox R.A.: Environmental sensitivities: Prevalence of major symptoms in a referral Centre: The Nova Scotia Environmental Sensitivities Research Centre Study. Environmental Health Perspectives. 109(2): 161-5.
5. McKeown-Eyssen G, Marshall L, Ross G, Kronki M, Sokoloff E 1994. The University of Toronto Health Survey on Environmental Hypersensitivity. A Report to the Ontario Ministry of Health, Toronto, Canada: Ontario Ministry of Public Health Publications.
6. McKeown-Eyssen GE, Sokoloff ER, Jazmaji V, Marshall LM, Baines CJ 2000. Reproducibility of the University of Toronto self-administered questionnaire used to assess environmental sensitivity. American Journal of Epidemiology 151(12), 1216-1222.

Objectives & Notes

Martha Stark, M.D.

Date of talk: Sunday, June 11, 2006, 10:30am

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Training:

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Current Faculty Appointments:	Center for Psychoanalytic Studies, Massachusetts General Hospital, Harvard Medical School; Massachusetts Mental Health Center, Harvard Medical School; Faculty, Boston Psychoanalytic Institute; Faculty, Massachusetts Institute for Psychoanalysis
Medical School/ University Attended	Harvard Medical School
Residency:	Adult Psychiatry Residency – The Cambridge Hospital, Cambridge, MA; Child Psychiatry Fellowship – Massachusetts Mental Health Center, Boston, MA; Psychoanalytic Training – Boston Psychoanalytic Institute, Boston, MA
Board Certifications:	American Association of Psychiatric Medicine
Other Information: (including titles of books or articles you have recently written):	“Working with Resistance”; “A Primer on Working with Resistance”; and “Modes of Therapeutic Action: Enhancement of Knowledge, Provision of Experience, and Engagement in Relationship”.

SPEECH TITLE: “Reversibility of Hypersensitivity and Degenerative Disease: Mechanisms”

At the end of this Presentation, the participant should be able to:

1. Understand the role of the ground regulation system in the maintenance of health.
2. Appreciate the importance of the “point of accumulation” in the body’s transitioning from a periodic to an aperiodic state.
3. Recognize the ways in which the living matrix is a complex system.

The above information was provided by the Speaker.

REVERSIBILITY OF HYPERSENSITIVITY AND DEGENERATIVE DISEASE: Mechanisms According to Dr. Rea

Martha Stark, M.D.
Sunday, June 11, 2006, 10:30am

My presentation is an effort to tease out some of the groundbreaking concepts presented by Dr. William Rea in his new book, to be entitled *The Reversibility of Chemical Sensitivity and Chronic Degenerative Disease*. In that book, Dr. Rea speaks to the pivotal role of the living matrix (variously described as the connective tissue matrix or the ground regulation system) – a master system that regulates and controls all the physiological processes and vital functions of the entire body. He distinguishes between messages that travel through the matrix by diffusion through the ground substance (the neurotransmitters of the nervous system, the hormones of the endocrine system, the cytokines of the immune system) and messages that travel in the matrix itself by electronic conduction along the protein backbone of the PG/GAGs or by the hopping of protons in the layers of water bound to these sugar-protein complexes.

More generally, Dr. Rea develops the idea that the living matrix, by virtue of its interconnectedness with every cell in the body and its ability to transmit energy and information at high speed to every part of the organism, is responsible for the health and vitality of the body. In fact, the health of the cells is dependent upon the health of the living matrix in which they are embedded and bathed. The matrix can function as a global communication system because it is a highly ordered array of molecules (primarily water, proteins, and glycoproteins) closely packed and tightly organized with a high degree of regularity in crystal-like lattice structures – a remarkable arrangement that accounts in large part for the adaptability and resilience of the living system.

Dr. Rea advances the idea that the living matrix is best understood by studying its parts in interaction (a holistic approach) as opposed to studying its parts in isolation (a reductionistic approach) and that, as such, the living matrix is truly a whole that is greater than the sum of its parts. By the same token, Dr. Rea conceives of the living matrix as an energetically open, self-organizing, complex system that is subject to the laws of deterministic chaos – meaning, amongst other things, that the matrix allows for the flux of energy and information through it, is regulated by feedback loops, is able to organize ever more complex structures as emergent properties of the system as a whole, and is always part predictable and part random in terms of its evolution.

Periodicity and reversibility are emergent properties of the living matrix. Dr. Rea's vision is ultimately a hopeful one because he highlights that there are ways to intervene that will support the adaptability and the resilience of the matrix and, therefore, the restoration of health. Such treatments include (1) detoxification (to decrease the "bad"), (2) replenishment (to increase the "good"), and (3) recovery of order and structural integrity so as to restore regulatory capacity, synchronization, and balance.

It is only the cumulative impact of unusual or extreme stress over time and a system that has lost its resilience and capacity to adapt (because its nutrient, adaptation, and energetic reserves have become depleted) that may reach the point of no return, at which stage the system will collapse into a chronic progressive degenerative state – with structural damage (on the level of the cell) replacing the earlier functional impairment (at the level of the matrix). At this point, health is irretrievable because order and organization can no longer be restored.

Emoto, Masaru (2004). *The Hidden Messages in Water*. Hillsboro, OR: Beyond Words Publishing.

Heine, Hartmut (2000). *Homotoxicology and Ground Regulation System (GRS)*. Baden-Baden, Germany: Aurelia-Verlag.

Oschman, James (2000). *Energy Medicine: The Scientific Basis*. New York, NY: Churchill Livingstone.

Oschman, James (2003). *Energy Medicine in Therapeutics and Human Performance*. New York, NY: Butterworth Heinemann.

Pischinger, Alfred (1991). *Matrix and Matrix Regulation: Basis for a Holistic Theory in Medicine*. Brussels, Belgium: Haug International.

Objectives & Notes

Nancy A. Didriksen, Ph.D.

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Current Job Description:	Private Practice, Richardson, TX, evaluating and treating patients with chemical/environmental sensitivities and related illnesses.
Current Faculty Appointments:	Adjunct Professor of Psychology, University of North Texas
Medical School/ University Attended	University of North Texas
Internship:	Environmental Control Unit, Northeast Community Hospital, Bedford, TX

SPEECH TITLE: “**Neurocognitive Effects of Toxigenic Molds**”

At the end of this Presentation, the participant should be able to:

1. Describe the neuropsychological test results of patients reporting primary exposures to toxigenic molds.
2. Describe the types of neurocognitive deficits found most frequently in mold-exposed individuals.
3. Discuss the physical, psychological, and neurocognitive symptoms endorsed most frequently on the Checklists by individuals exposed to toxigenic mold and whether the reported neurocognitive symptoms are consistent with neuropsychological findings.
4. Discuss confounding variables in data analysis and their importance in disability and litigation issues.
5. Describe future research regarding the long-term sequelae of toxigenic mold exposure.

The above information was provided by the Speaker.

Neurocognitive Effects of Toxigenic Molds

Nancy A. Didriksen, Ph.D.

Significant controversy continues regarding cognitive impairment resulting from exposure to toxigenic molds. Several investigators (Baldo et. al. [2002], Warden & Cantor [2004], Crago et. al. [2003], and Kilburn [2003]) have found a variety of neurocognitive deficits associated with mold exposure. Findings are consistent when similar measures have been utilized and have been compared with the neurocognitive deficits found in mild traumatic brain injury patients.

The neurocognitive data of 41 patients reporting primary mold exposures were previously examined by this investigator. The present study has examined the data of 60 mold-exposed individuals (mean age 49.18, mean educational level 15.67) referred to this office for neuropsychological assessment, primarily associated with disability and litigation issues.

More recent assessments primarily utilized the Halstead-Reitan Neuropsychological Test Battery, the Wechsler scales, and the Test of Memory Malingering as a measure of motivation and effort. Patients were also administered a clinical interview and mental status examination and completed a Physical, Psychological, and Neurocognitive Symptom Checklist as part of the assessment.

The most frequently reported symptoms remain quite similar to those reported in the earlier study. These include fatigue, low energy, weakness, difficulty remaining asleep, skin problems, headaches, sinus discomfort, aches and pains, eye problems, “sick all over”, present performance inferior to prior performance or level of functioning, “This is not me”, “cloudy, foggy, spacey”, difficulty getting started in the morning, worry about bodily dysfunction, tension, difficulty setting and reaching goals, feels like “insides are racing”, decreased coping ability, decreased attention, concentration, memory, and comprehension, naming and word-finding problems, intellectual inefficiency, distractibility, loss of organizational skills, and difficulty with decision making.

Neurocognitive test data yielded the following results: Wechsler Adult Intelligence Scale-Revised Full Scale IQ scores fell within normal limits, ranging from average to very superior, with ten percent of patients falling in a low-average range on the Performance measures. Six patients were evaluated using the Wechsler Adult Intelligence Scale-III. Two patients scored in a low-average range and none scored in a very superior range. Scores on the Wechsler Memory Scale-III fell at approximately the population mean (50th percentile) with no significant differences between verbal and visual memory or between immediate, delayed, and working memory.

Thirty-four percent of the thirty-eight patients to whom the entire Halstead-Reitan Neuropsychological Test Battery was administered scored within normal limits, overall. Forty-five percent of the patients demonstrated mild impairment and twenty-one percent scored in a moderately impaired range. Only eighteen percent had an Impairment Index within normal limits. Greatest impairment was found on measures of higher cortical functions, consistent with the prior study as well as with data analyses of other types of neurotoxic exposures.

Age, sex, educational, and racial norms were utilized in addition to the standardization group norms to determine whether a decrement in functioning had occurred. Additionally, prior levels of educational and occupational performance were considered in the interpretation of test results, particularly important for disability determination. All patients to whom a malingering test had been administered scored within normal limits with one exception.

Many factors, including time elapsed from exposure to evaluation (0-67 months), duration of exposure (less than 1 month to 13+ years), documentation of the mold exposure, other environmental exposures/sensitivities, motivation of the individual, reactions to environmental incitants at the time of evaluation, head trauma and/or other neurological conditions, effects of medications, metabolic disorders, past or present drug and alcohol abuse, and psychological factors were considered in the interpretation of individual test data to determine whether the observed deficits were due primarily to mold exposure. These factors are particularly important for litigation issues.

Conclusions:

The neuropsychological test data of the 60 mold-exposed individuals in the present study are consistent with the pattern of deficits found in patients exposed to other neurotoxins (solvents, pesticides, metals), consistent with self-report of dysfunction, and consistent with the findings of other investigators. Results suggest that the molds to which these patients were exposed have neurotoxic properties. However, deficits do not appear to be as severe as those observed with other types of neurotoxic exposures.

Frequency, intensity, and duration of exposure must be considered as well as individual susceptibility, sensitivity of the tests administered, and the relatively small sample size. It is possible that the effects of toxigenic molds are less severe than other neurotoxins.

Further research is necessary regarding the neurotoxic effects of specific molds. Factors to be considered include the types of individuals who may have greater susceptibility to their neurotoxic effects on cognition, conditions which appear to enhance the adverse effects of mold exposure, interventions which will most effectively treat the adverse effects of toxigenic mold exposure, and whether the illness resulting from mold exposure may be a permanent condition.

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Objectives & Notes

William J. Meggs, M.D., Ph.D.

Date of talk: Sunday, June 11, 2006, 11:30am

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Other Information: (including titles of books or articles you have recently written):	Book: The Inflammation Cure, McGraw-Hill (2003)/ Fallacies in Refutation of Causality, published in Clinical Toxicology, 2005. Sustained oxygenation without ventilation, published in Amer. Journal Emergency Medicine in 2005. Pressure Immobilization Bandages delay of toxicity of coral snake bites, published in Annuals of Emergency Medicine, 2005.

SPEECH TITLE: **“Toxicity of Chronic Low-Dose Organophosphate Exposures”**

At the end of this Presentation, the participant should be able to:

1. To know the history of organophosphate compounds and their acute toxicity
2. To know the mechanisms of action of organophosphate compounds
3. To know the effects of chronic exposures to organophosphate compounds

The above information was provided by the Speaker.

Toxicity of Chronic Low-Dose Organophosphate Exposures

William J. Meggs, M.D., Ph.D.

Abstract

There are ubiquitous exposures to organophosphate insecticides world wide. While the acute effects of exposure are well known, effects of chronic exposures that do not produce acute toxicity are not as well defined. Data from various sources indicate that chronic exposures to low doses of organophosphate compounds can produce adverse neurological, endocrinological, immune, and psychiatric effects. Recent experimental work suggests that chronic exposure to organophosphates may be associated with abnormal weight gain, and that these compounds may play a role in the obesity epidemic.

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